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THE DIAGNOSIS AND TREATMENT OF ACUTE MALIGNANT OBSTRUCTION OF THE LARGE BOWEL*

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Recent reports on the treatment of carcinoma of the large bowel reveal excellent results in elective resection. These studies, however, pass lightly over one phase of the disease which is a frequent cause of disaster. I refer to acute obstruction of the colon due to carcinoma. Furthermore, this serious complication is infrequently discussed in its own right and often it is glossed over with brief statements to the effect that surgical decompression is the treatment of choice.

It is my impression, from a recent study[1] of 55 cases of acute intestinal obstruction of the colon due to carcinoma, that there is no unanimity of opinion concerning the manner in which this condition should be handled.

The 55 cases which make up this series were collected from Charity Hospital and Touro Infirmary, in New Orleans. During the three year period ending December 31, 1945, 135 patients with carcinoma of the large bowel, exclusive of the rectum, were treated at Charity Hospital, 35 of whom (25.9 per cent) were completely obstructed. During the six year period ending December 31, 1946, 68 patients with carcinoma of the large bowel, exclusive of the rectum, were treated at Touro Infirmary, 20 of whom (29.4) per cent were completely obstructed.

In this series, 25 different surgeons were involved, one of whom treated eight of these patients, but 10 of whom were responsible for one patient each. As a result of the distribution of material among so many surgeons, there was no such uniformity of treatment as there would be, for instance, in a series of cases reported from a clinic such as Wangensteen's[2]. On the other hand, the high case fatality rate (almost 33 per cent) is probably more representative of the general mortality of acute malignant obstruction of the large bowel than is the far more favorable rate reported from clinics in which all cases are managed by the same general routine.

In this series, 52 of the 55 were operated on and eight different types of operative procedures were performed. In three other cases, conservative treatment was used entirely. Such a wide difference of opinion among surgeons in the same section of the country handling a similar condition, shows a lack of unanimity of opinion concerning this condition, and for this reason I believe that a closer study of this condition is merited.

GENERAL CONSIDERATIONS

There are certain considerations in acute malignant obstruction of the colon which set it apart as a definite disease entity and deserve special discussion.

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The fact that malignancy is the cause of obstruction immediately introduces the fact that the patient is in double jeopardy, both from the malignant disease and from a complication that is exceedingly serious in its own right. In obstruction due to non-malignant causes, the correction of the obstruction also corrects the cause of the obstruction. In acute malignant obstruction of the colon, the obstruction must be relieved first and resection is to be performed at a later date.

Carcinoma is by far the most common cause of acute obstruction of the large bowel. There is a general agreement that approximately 90 per cent of all obstructions of the large bowel are of malignant origin and that about one-third of all patients with carcinoma of the colon will eventually develop acute complete obstruction or such a degree of chronic incomplete obstruction that surgical decompression is necessary before resection can be performed. This series which I have studied, as already intimated, includes only the acute complete obstructions. Cases of intermittent or partial obstruction, even those which were submitted to surgical decompression prior to radical surgery, have not been included.

The incidence of acute colic obstruction in other series ranges from 35 per cent to as little as 5 per cent. Some series in which the incidence of obstruction is high include cases of subacute or chronic obstruction. Series in which the incidence is unusually low are from clinics in which little emergency surgery is done.

A second consideration, in acute malignant obstruction of the colon, is that the patients are most often in the older age group. This introduces complications not met with in younger persons, particularly degenerative diseases. Age itself, however, is not regarded as a contraindication to prompt surgical intervention. That age plays some part, however, is clear from the fact that in this series the average age of the group which survived was 53.4 years, against an average age of 55.8 years for the whole series and of 60.7 years for the group which did not survive.

The age range in this series was from 13 to 86 years; both patients at the extremes of life, incidentally, were numbered among the survivors. That 11 of the 55 patients were under 50 years of age and that two were under 30 years of age is an interesting commentary on the all too general belief that carcinoma is a disease of middle and late life. In this series, there were 25 females and 30 males. It is of interest that 24 of the 35 cases at Charity Hispital occurred in negroes, and it is also not surprising that 10 of the 12 deaths at that institution occurred in the same race.

A third important consideration is that the patient with obstruction of the colon due to malignant disease has been ill for a considerable period of time, even if, as happens occasionally, there has been no previous evidence of disease. For a variable length of time, before the patient becomes completely obstructed, the malignant growth has been slowly reducing the patient's vitality. Often, in malignancies of the right side of the colon, marked anemia is present. In this series, it was found that a large percentage of the patients had marked deficiencies in plasma proteins. Usually, the patient has been losing weight, and, as Wangensteen[2] has aptly expressed it, is "autocannibalistic."

Such patients are in marked contrast to those with acute small bowel obstruction, or with large bowel obstruction of non-malignant origin, who, as a general rule, are precipitated into their acute illness from a state of previous good health. When obstruction of the colon due to malignancy is not complete, the patient can be carefully prepared for operation by replacement of the carbohydrate, protein, vitamin and hemic deficiencies, as well as by chemotherapy and bowel irrigation. Such preparation is a time-consuming process, and the patient with acute obstruction has no time whatsoever for such procedures. In most of these cases, therefore, the debility caused by the primary disease is added to the infirmities of age and the seriousness

of the acute obstruction, and the surgical risk is thereby compounded.

PHYSIOLOGY AND PATHOLOGY

Acute obstruction of the colon differs from acute obstruction of the small bowel in that the former is most often of the closed loop type, the growth forming the distal occlusion and the ileocecal valve the proximal occlusion. When once this loop is closed, distention of the colon is rapid, because each peristaltic wave forces the contents of the ileum into the cecum. The competency of the ileocecal valve prevents regurgitation from the cecum into the ileum. As distention increases, edema, thrombosis, ulceration and infection occur in the bowel wall until eventually, if surgical relief is not achieved, perforation will occur.

In this particular series of cases, the statement often made that perforation usually occurs in the cecum, because of its relatively thin wall and distensibility, is not necessarily true. Perforation can occur at any point above the lesion. Of the eight proved perforations in these 55 cases, only two developed in the cecum. Four of the remaining perforations were in the sigmoid, in two instances as the result of traumatic manipulation at operation. One perforation occurred in the descending colon, and one occurred at the hepatic flexure.

Wangensteen[5] and his associates have made important contributions, both experimentally and clinically, to the knowledge of the role of the ileocecal valve in obstruction of the large intestine. Its competency is variable. By correlation of roentgenologic observations and findings at operation, Dennis[3] determined that this valve was competent in 61 percent of 53 cases of acute colic obstruction. However, it is to be emphasized that attempts to differentiate clinically between a competent and an incompetent ileocecal valve are likely to delay surgical relief of the obstruction and may greatly jeopardize the patient's chances of recovery.

In every case in this series, the malignant growth was an adenocarcinoma, and in the great majority of cases it was of the annular type, which readily gives rise to obstruction because it constricts the lumen of the bowel. In the few cases in which the growths showed gross fungating or polypoid characteristics, they also showed a marked tendency to infiltrate the wall of the colon and encircle the lumen, thus giving rise to obstruction.

LOCATION OF OBSTRUCTION

The exact location of an acute obstruction of the colon is an extremely important consideration from the standpoint of diagnosis and subsequent therapy. In this series, 31 of the 45 cases of obstruction occurred in the left colon. The same predominance of left sided obstruction is noted in all other reported series and can readily be explained.

The lumen of the left colon is generally smaller than the lumen of the right colon. Its walls are thicker and less distensible than the walls of the right colon. The fecal contents, which are more solid on the left than on the right, easily traumatize the malignant growth, causing secondary infection with ulceration, edema and swelling. Finally, the annuar type of adenocarcinoma, which by its very nature readily gives rise to obstruction, is somewhat more frequent on the left side than on the right.

The fact, however, that 14 cases of acute malignant obstruction did occur on the right side of the colon is important to note. This contradicts a rather widespread belief that such a condition rarely occurs.

CLINICAL PICTURE

In cases of acute malignant obstruction of the colon, the history can often be divided into two distinct periods, one of long term, indefinite symptoms and the other of acute symptoms. However, very often the period of indefinite symptoms will be entirely absent or may blend gradually into the symptoms of acute complete obstruction.

In the typical case, the original period was characterized by an insidious onset and a triad of symptoms including: (1) altered bowel function; (2) melena, and (3) abdominal pain, which, until the onset of acute symptoms, was mild and transient.

Some patients had had previous mild acute attacks suggestive of incomplete obstruction. In a small number of cases, the development of the acute obstruction seemed to be the first manifestation of the disease.

The signs and symptoms of acute obstruction of the lage bowel in this series were as follows:

1. An inability to defecate or to expel flatus. In many cases, the patients complained of a great desire to defecate. This probably explains why so many resorted to purgation before consulting a physician.

2. Cramping abdominal pain was present in practically every case. It was spasmodic and often extremely severe. Visible peristalsis was frequently present.

3. Thirty-nine of the 55 patients vomited during the acute attacks and four others complained of nausea but did not vomit. The remaining 12 patients did not complain of either nausea or vomiting. Wangensteen has commented repeatedly on the possibility that acute obstruction of the large bowel can occur without vomiting. This, in his opinion, is due to the closed loop character of the obstruction and the absence of distention or the presence of only minimal distention of the small intestine.

4. Abdominal distention was usually pronounced in acute malignant obstruction of the colon, except in cases in which a closed loop type of obstruction was present due to a colic neoplasm.

5. Abdominal tenderness was a frequent finding and was often localized in the lower right quadrant. This localization of tenderness is explained by the presence in that area of a distended cecum, which sometimes could be palpated as a tender mass, even when the malignancy was located in the left colon. Wangensteen has pointed out that this is the result of the thinness of the wall of the cecum and its greater distensibility as compared to the rest of the colon.

6. The presence of an intra-abdominal mass, which is the malignant tumor itself. Palpable growths were more often found in malignancies of the right colon, but occasionally the growths on the left side could be palpated.

7. A low grade fever, which was present in a large number of cases, was probably the result of ulceration and infection of the wall of the colon proximal to the neoplasm.

The pronounced chemical changes which ensue so rapidly in obstruction of the small intestine are not present in obstruction of the colon. In this series, the only consistent abnormal laboratory findings were a slight leukocytosis, increased polymorphonuclear leukocytes, varying degrees of anemia (especially in carcinoma of the right colon), and a persistent reduction in serum protein levels.

DIAGNOSIS

In the majority of cases in this series the diagnosis was correct, or obstruction of the large intestine due to malignancy was listed as an alternative diagnosis. Small bowel obstruction was the only diagnosis or alternative diagnosis in six cases, volvulus in four, acute appendicitis in three, acute cholecystitis in two, diverticulitis or paralytic ileus in one case each.

An analysis of the cases incorrectly diagnosed as small bowel obstruction is instructive. In two patients the obstruction was due to carcinoma of the cecum. It is actually impossible to differentiate obstruction of the ileocecal junction due to this cause from any other type of low ileal obstruction by either clinical or roentgenological methods. Such a diagnostic error can be important, however, because some surgeons may be tempted to treat lower small bowel obstruction by intubation methods, which, as will be shown later, are definitely contraindicated in malignant obstruction of the colon. In the other cases in which an incorrect diagnosis of small bowel obstruction was made, the error was due entirely to a failure correctly to interpret the flat films or a failure to take any x-rays whatsoever.

The frequency of localization of symptoms and signs in the right lower quadrant, which has already been commented on, is the obvious explanation of the diagnosis of acute appendicitis in malignant obstruction of the large bowel. This mistake is most

likely to occur when the obstruction is on the right side of the colon, though it can also occur when left-sided lesions are present. The latter is explained by the marked distensibility of the cecum.

A low-grade fever, leukocytosis, an increase in the polymorphonuclear leukocytes, which all may be present in malignant obstruction of the colon, are further reasons for a possible incorrect diagnosis of acute appendicitis. In this connection, the following case is of special interest.

A 32 year old woman had had recurrent attacks of pain in the right lower quadrant, nausea and vomiting for six months. A few days after the latest attack, she was operated on through a right rectus incision, with a diagnosis of acute appendicitis, which was not verified by the surgeon at operation or by the pathologist. She returned to the hospital two weeks after her discharge, with the same symptoms as before operation and with marked abdominal distention. A barium enema at this time permitted a diagnosis of obstruction due to carcinoma of the sigmoid colon, which was relieved by colostomy.

Carcinoma of the colon introduces a reverse diagnostic problem in acute appendicitis. In Boyce's[6] material on acute appendicitis, collected over an almost 16 year period from Charity Hospital, three patients over 40 years of age died without operation from appendiceal peritonitis because the diagnosis of carcinoma of the colon had been made. In six other cases, operation was delayed because of a similar mistake in diagnosis. One of these cases ended fatally, because the patient was being prepared for surgery with the idea of a colectomy being performed.

It is of interest that is some of the cases in which the right side of the colon was resected for obstructing malignancy, the pathologist reported the appendix to be acutely inflamed. These pathologic changes are the result of back pressure, which causes inflammatory changes in the wall of the appendix, just as in the wall of the cecum and the colon proximal to the obstructing growth.

The incorrect diagnoses are significant from the standpoint of their responsibility for a delay in surgical therapy. A patient with obstruction due to a paralytic ileus is not likely to be operated on at all. A patient with cholecystitis may or may not be operated on. Many surgeons may not operate immediately for presumed simple obstruction of the lower ileum.

On the other hand, a patient with volvulus and a patient with acute appendicitis is usually operated on without delay and these particular errors, fortunately, were in the category of conditions which demand prompt operation. However, it is to be emphasized that in dealing with acute malignant obstruction of the colon the importance of an exact diagnosis beforehand cannot be too strongly emphasized. Abdominal exploration in such cases definitely places the patient in a more precarious situation.

In this series, the fact was brought out that the majority of patients had had abdominal symptoms for relatively long periods of time before seeking medical advice. It is true that unless an individual seeks medical aid, medical aid cannot be given to him. The importance of early diagnosis in cancer and the value of our present campaigns for increasing the layman's knowledge of the disease is well brought out.

However, on the other hand, it is ironic and tragic that eight patients in this series did seek medical aid, at intervals varying from one to eight months before they entered the hospital with acute malignant obstruction. The patients all consulted their physicians because of abdominal symptoms, particularly vague abdominal pain. Three patients were told by their physicians to take purgatives. One was treated for seven months for dysentery, which he might or might not have had also. The presence of dysentery bacilli or ameba in the stools does not rule out the presence of a concurrent neoplasm. One patient was told that he had a gastric ulcer and another was told that her abdominal symptoms were due

to a gynecological disturbance. The conclusion is inevitable that in none of these cases was the slightest attempt made to study the patient exhaustively and to arrive at an accurate diagnosis, though most of them presented the well-known triad of altered bowel function, melena and cramping abdominal pain.

ROENTGENOLOGIC DIAGNOSIS

X-ray studies of the abdomen, both by flat plate and barium enema, are of extreme importance in making a correct diagnosis in acute malignant obstruction of the colon.

In this series, flat plates were made in practically all cases with generally uniform findings, in the form or marked distention of the colon up to the site of growth. Distention of the small intestine was present in a larger proportion of cases than the writings of Wangensteen might indicate.

The presence of distention of the small intestine can be explained in several ways. When the ileocecal valve is incompetent, naturally the small intestine takes part in the distention. Even when the valve is competent, and a so-called closed loop obstruction is present, it is logical to assume that pressure in the cecum will eventually reach such a point that the valve cannot open in response to ileal peristalsis and small bowel distention will eventually occur.

Furthermore, when pronounced distention of the large bowel is present, plus extensive inflammatory changes in the wall of the colon, some degree of paralytic ileus is likely to take place in the small bowel. The chief importance of the presence of small bowel distention in association with large bowel obstruction is that the primary source of the occlusion may be overlooked and decompression by non-surgical methods may be substituted for surgical decompression.

It should also be noted that carcinoma of the cecum with involvement of the ileocecal valve causing obstruction presents roentgenologic findings distinctly different from those observed in occlusion elsewhere in the large bowel. In such cases there is no distention of the colon and the findings are typical of low ileal obstruction.

The plain film should be taken with the patient in the upright position. Additional information often can be gained from the upright film that is not evident from a film taken with the patient lying down.

An immediate barium enema should always be done in cases in which malignant occlusion of the colon is suspected. In the series which we have reviewed, this method of examination was used before operation in only 15 of the 55 patients. It seems significant that it was not employed in any of the incorrectly diagnosed cases. Even if the diagnosis of colonic obstruction can be definitely made without the assistance of the roentgenogram, the barium enema is necessary for exact localization of the growth, so that the incision can be correctly placed in reference to it and that exploration at operation can be reduced to a minimum. Furthermore, if the growth is accurately located before operation, the exact operative procedure can be decided upon beforehand and can be done rapidly with a minimum of manipulation.

Certain precautions, of course, are necessary. The barium must be introduced gently and slowly and under low pressure. Care must be taken that it is not forced past the site of obstruction for two reasons. In the first place, the exertion of undue pressure can readily cause perforation of a friable, secondarily infected intestinal wall, infiltrated with malignancy, which lacks the distensible properties of the normal colon. In the second place, if barium is forced past an incompletely obstructing growth, the possibility exists that the obstruction may be completed. If barium remains in the right side of the colon for even a short period, the normal absorptive properties of the colon soon reduce the barium to an inspissated mass which can completely block the intestinal lumen. A case of this kind, which is not included in this series, recently came under my observation.

Useful as barium is in the diagnosis of obstruction of the large bowel when given as an enema, it is extremely dangerous when given by mouth in any type of intes-

tinal obstruction. An incomplete obstruction may be converted into a complete one, the presence of the opaque medium in the bowel can greatly complicate the operation, and the risk of perforation is greatly increased. In one case in this series, in which conservative treatment was used, a dual perforation of the bowel in the transverse colon and sigmoid, and fatal generalized peritonitis followed immediately on the use of barium by mouth as a diagnostic measure.

TREATMENT

That there is a lack of uniformity of opinion concerning the handling of acute malignant obstruction of the colon is evident from this series which I have reviewed. Three of the patients were treated conservatively, and eight different types of operative procedures were performed in the other patients.

There is no question but that prompt surgical decompression of the colon is indicated in acute malignant obstruction of the colon regardless of the exact site of the lesion. This opinion is also that of Wangensteen,[5] Dennis,[3] Rankin,[4] and others. Conservative therapy and attempts at intestinal intubation have no place whatsoever in the treatment of acute malignant obstruction of the colon.

Conservative therapy: In three patients in this series, conservative treatment was used completely. Two of these cases terminated fatally in the hospital. Necropsy examination in the first, a carcinoma of the sigmoid, revealed generalized peritonitis following perforation of the bowel at the hepatic flexure. In the second, a carcinoma at the hepatic flexure, the patient died suddenly after 15 days of hospitalization, with signs and symptoms suggestive of perforation of the cecum. The fact that one of these three patients was able to overcome the obstruction after conservative treatment is certainly no indication that this is the correct way to handle such cases. The physician who elects to treat complete malignant obstruction of the large intestine by conservative measures is indulging in wishful thinking. As Pfeiffer and Martin[7] have put it, "relief by this means may be hoped for but should not be expected."

In patients in whom the obstruction of the colon due to carcinoma is not complete, close observation must be the policy. If there is any question of the obstruction becoming complete, surgical decompression should be undertaken as soon as possible because of the possibility of closed loop obstruction and perforation of the colon.

Due to the fact that the onset of complete obstruction is occasionally insidious, it is evident that surgical decompression is often unnecessarily delayed while the patient is in the hospital. In seven of the 15 patients who died, operation was delayed from two to 14 days. In eight of the 37 patients who survived operation, surgical intervention was delayed from 10 to 34 days. The obstruction was temporarily relieved and the patient treated from 10 and for 34 days, respectively, but in each instance it recurred as soon as treatment was discontinued and surgical decompression had to be resorted to.

In at least three cases in this series, there seems little doubt that while an incomplete obstruction was being treated conservatively, the obstruction became complete. In another case, the patient was being prepared for elective colectomy for carcinoma of the cecum by the use of castor oil. In the course of the preparation, she developed signs and symptoms of acute obstruction, for which ileostomy was performed. Death occurred 48 hours later, and necropsy revealed an obstructing carcinoma of the cecum which had perforated.

That intestinal intubation with the Miller-Abbott tube is of permanent, curative value in any type of obstruction of the colon is not now generally believed. In this series, it was practiced in 20 cases, but the tube entered the small intestine in only three cases, two of which terminated fatally. Although it is entirely permissible to use the tube in conjunction with surgical decompression, the use of intestinal intubation is certainly not a substitute for surgical decompression in such cases. When the ileocecal valve is competent, the colon is

unable to empty its contents in the direction of the ileum, although the ileum itself is able to evacuate itself into the cecum, with a resulting increase in a distention which is already present. The length of time necessary for a Miller-Abbott tube to traverse the distance from the stomach to the ileocecal junction is entirely too long to delay decompression of the large bowel proximal to the growth.

The tube is useful only when it can be passed with a reasonable degree of speed (which can be increased by the use of mercury in the balloon, as suggested by Harris[8]) and thereby can deflate edematous, distended loops of small intestine which may be present and prevent further influx of the contents of the small intestine into the cecum. It usually has no other influence on a large bowel distention and such relief if secured may be highly misleading, thus leading to further delay.

These remarks, of course, have nothing to do with the use of intubation after operation, which should be routine in all surgery of the large bowel. Simple duodenal suction is usually all that is necessary, but the Miller-Abbott tube should be employed if small bowel distention is pronounced. One criticism which can be advanced against the postoperative treatment applied in this series was that in many cases the impression seems to have occurred that because the Miller-Abbott tube was not effective before operation, it would also be of no value after operation and was therefore removed.

RESECTION IN THE FACE OF OBSTRUCTION

In this series, immediate resection was carried out 10 times, with a mortality of 50 per cent. Five resections were done for carcinoma of the sigmoid, with two deaths; two in obstruction of the cecum, with two deaths; and once each in carcinoma of the hepatic and splenic flexures and of the right transverse colon. The patient with carcinoma of the splenic flexure also died.

This is a prohibitive mortality, and the performance of resection in the face of acute obstruction of the colon reveals a fundamental misconception of the pathologic background of the disease. As already intimated, there are two distinct problems to be dealt with. One, which is urgent and immediate, is the relief of the obstruction before the viability of the intestinal wall is irreversibly damaged by impairment of circulation. The second problem, which is not immediately urgent, is the removal of the malignant tumor. This should be dealt with at subsequent operation after the obstruction has been overcome.

When resection is done in the face of obstruction, all circumstances are against success. The condition of the patient is frequently very poor. The blood supply to the intestinal wall is impaired. The mucosa is ulcerated and infection of the bowel wall is present. The patient is dehydrated, starved and exhausted, and he may show impairment of the vascular system.

Immediate resection is certainly a temptation which always has to be faced when operating on such cases. It seems highly significant that the only patient in the group of fatal cases who was considered to be a good risk before operation lost that advantage when immediate resection was attempted in the face of obstruction. Two patients in the group of survivors who presented fair risks when they were first seen also lost that advantage when immediate resection was attempted. The pathologist reported that in these two instances the growth had been incompletely removed.

Resection was done as a subsequent operation in 22 of 32 patients who had surgical decompression only at the first operation. Of these 22 patients, there was only one fatality, which was the result of massive atelectasis. Had resection been attempted in these cases at the first operation, it seems fair to assume, on the basis of five deaths in the 10 cases in which resection was done in the face of obstruction, that a similarly prohibitive mortality would have occurred.

SURGICAL DECOMPRESSION

The type of surgical decompression which should be used depends on the location of the obstruction. For this reason, definite localization of the growth by means

of the barium enema before operation is indicated.

In acute malignant obstruction of the left colon (left half of the transverse colon, descending and sigmoid colon), the choice of operation lies between cecostomy and transverse colostomy.

Cecostomy was done in 24 patients in this series, with six fatalities. That cecostomy is frequently a life-saving measure and is a simple operative procedure there is no doubt. Another advantage of cecostomy is that very often it closes spontaneously.

However, there are numerous disadvantages to cecostomy in acute malignant obstruction of the colon. Aseptic decompression is seldom possible. Drainage of the bowel contents frequently occurs directly onto the skin. The operation may be difficult and dangerous when the bowel wall is thin and distended. Exteriorization of an intact segment of the cecal wall is often impossible and decompression is usually accomplished at the expense of some soiling. Decompression is less effective than when colostomy is performed. Defunctionalization is not achieved. Infection of the wound is common and wound dehiscence is not infrequent. If spontaneous closure does not occur, surgical closure may be difficult.

In my opinion, the operation of choice in acute obstructing malignancy of the left colon is a simple loop colostomy of the right side of the transverse colon. In this series, it was performed in only four patients.

Such an operative procedure has numerous advantages. The intestinal opening is considerably removed from incisions used for subsequent resection of left-sided growths. The operation is easily performed, for the transverse colon is very mobile and easily delivered. If the operation is performed through a small transverse incision, extrusion of the distended bowel is practically spontaneous. This is not as easily accomplished through a vertical incision, because of the twisting of the bowel on its axis. The use of a glass or plastic rod to support the bowel eliminates the necessity for suturing the colon to the layers of the abdominal wall.

Immediate decompression is obtained by placing catheters in both loops of the colostomy for 48 hours before the bowel is actually severed.

This procedure is further simplified by the adoption of Wangensteen's[5] suggestion that during roentgenologic examination a small opaque object be placed over the umbilicus, for later identification of the exact location of the transverse colon. Incision can then be placed directly over the colon and manipulation reduced to a minimum.

When transverse colostomy is done, defunctionalization of the bowel is almost complete. The patient can be ambulated rapidly and can usually consume his usual diet. The material which accumulates in the distal colon can be easily evacuated by means of irrigation.

Surgical closure is necessary in such colostomies, but is not particularly difficult. End-to-end closure can readily be performed after adequate chemotherapy, and the complications and difficulties of spur formation and spur crushing are avoided.

Good results of the simple loop transverse colostomy are evident in such a report as Fallis'[11] which shows 52 such operations for obstruction of the left colon, with only two deaths.

Devine was the first to suggest the utilization of the right half of the transverse colon as the site of a temporary colostomy for lesions of the left colon. A special type of procedure was prescribed and such colostomies were done in three patients in this series. Defunctionalization is complete and satisfactory by this method, but in my opinion this procedure is unduly complicated and has no advantages over a simple loop colostomy in acute left colic obstruction.

In this series, three colostomies of the sigmoid colon were done directly above the site of the growth. Such a procedure is not desirable because of the resulting fixation of the left side of the colon and technical difficulties involved in subsequent resection. There is a uniformity of opinion, among those who have studied this condi-

tion, that some type of right-sided decompression (either cecostomy or colostomy) should be done in left-sided obstruction and that no manipulation or exploration of the lesion is indicated.

Right-sided malignant obstruction of the large bowel presents a different problem from that of obstruction of the left colon. The choice of operative procedure for right-sided malignant obstruction lies between cecostomy, ileostomy, or ileotransverse colostomy. Cecostomy, as has previously been mentioned, is an adequate procedure for decompression of the colon and is particularly useful in obstructing carcinomas which occur either in the ascending colon, hepatic flexure, or right side of the transverse colon. Naturally, it is of no use and should not be performed in obstructing carcinoma of the cecum or ileocecal region. One disadvantage of cecostomy is that the right side of the colon is fixed to the abdominal wall and subsequent resection may be rendered somewhat difficult.

As Rankin[4] suggested some 20 years ago, and as Wangensteen[5] also advocates, it is a feasible plan in obstructing carcinoma of the right colon to open the terminal ileum and insert the catheter through the ileocecal valve into the cecum and perform a catheter ileostomy. Such a procedure would perform essentially the same degree of decompression as a cecostomy or appendicostomy. Ileostomy was performed in only two patients in this series, and both died. However, both patients were moribund when operated upon, and the value of the procedure cannot be judged from these two isolated cases.

In right-sided obstruction, ileotransverse colostomy, producing a short circuit around the growth, has certain advantages over enterostomy. At subsequent operation there is no enterostomy to be dealt with and there is no fixation of the right side of the colon. It is undoubtedly a safe and useful procedure in selected cases, in which the intestine proximal to the growth is not markedly distended and in which the ileocecal valve seems to be incompetent. However, it is more time-consuming than a simple enterostomy. This is a serious consideration in gravely ill patients.

On the other hand, if the ileocecal valve is competent, the closed loop may still persist after the performance of ileotransverse colostomy. Therefore, some type of decompression may be necessary in addition to the ileotransverse colostomy. An excellent procedure is to completely sever the ileum and bring the distal end out as a mucous fistula.

In the six persons in which ileo-transverse colostomy was done, an ileostomy was utilized as an adjunct in one case, while in another case the surgeon performed appendectomy, decompressed the cecum via the appendiceal stump and then closed the stump.

OTHER THERAPEUTIC CONSIDERATION

Anesthesia: My personal preference, in such cases in which abdominal distention is marked, is for the use of spinal anesthesia. The maximum of relaxation is obtained, and excessive respiratory movements, which are likely to be present during general anesthesia, are not encountered. If the patient is unduly apprehensive, nitrous oxide, ethylene or sodium pentothal can be added.

The frequency with which patients come to the operating table with gastric, duodenal or Miller-Abbott tubes in situ may complicate the use of general anesthesia, but the latter is perfectly feasible if spinal anesthesia is contra-indicated and if a competent anesthetist is available.

In this particular series, competent physician-anesthetists were available in all cases, and in no instance did the anesthesia *per se* play any part in the fatal outcome.

Chemotherapy: Chemotherapeutic and antibiotic agents were used in only 15 of the 55 cases in this series. This number included five of the 18 fatal cases. Whether a wider use of these new agents would have reduced the number of fatalities, it is impossible to say. However, it is my opinion that the unwise surgical procedures which were used in some of the cases and long periods of temporizing measures introduced

in others were really the important factors in the mortality and that the addition of chemotherapy would have had little effect.

My own opinion is that sound surgery is of preeminent importance in the condition herein discussed, but the adjunct use of penicillin in massive doses postoperatively is justified. The method recently described by Crile[12] for appendiceal peritonitis, in which doses of 100,000 units of penicillin every two hours are given intramuscularly, can readily be applied in acute malignant obstruction of the colon in which perforation may occur.

Suture material: In this particular series, the four incidents of wound dehiscence are perhaps the proportion to be expected in a series of cases of malignant obstruction due to carcinoma of the colon. No particular conclusions can be drawn as to why these occurred, because of the numerous varieties of suture material which were employed by the 25 surgeons involved. The level of protein deficiencies in these patients was, of course, important.

My personal preference is for the use of fine cotton for the closure of abdominal wounds, particularly in such cases as these. I also believe that the incidence of wound infection can be markedly reduced by the use of secondary wound closure in all cases in which contamination has occurred.

MORTALITY

Three previous series of cases of intestinal obstruction studied at New Orleans Charity Hospital invite comparison with this series. In the first, reported by the late C. Jeff Miller[13] in 1929, there were nine cases of malignant obstruction of the large bowel, with seven deaths. In the second, reported by Moss[14] in 1934, there were four instances of this type of obstruction, with two deaths. In the third, reported by Boyce[15] in 1937, there were 11 such cases with nine deaths. Case fatality rates for these early series are higher than those for the series which I have reported (32.7 per cent), but this latest series also bears out Rankin's gloomy prophecy that "even with the simplest possible surgery the case fatality rate for this type of obstruction will be at least 30 per cent, and the rate will rise proportionately as more extensive surgery is done."

CAUSE OF MORTALITY

It is evident from a study of this series, that the high mortality rate can be attributed to the following: (1) attempts at conservative therapy; (2) resection in the face of acute obstruction; (3) delay of patients seeking medical attention; (4) delay in diagnosis after the patient is seen by a physician; (5) attempts at surgical exploration at operation with resulting perforation of the growth due to trauma; (6) the use of purgatives; (7) the use of barium by mouth; (8) the patient's age; (9) associated systematic disease; (10) debilitation due to metastasis.

SUMMARY

1. Fifty-five cases of acute obstruction of the colon due to carcinoma have been analyzed.

2. A lack of uniformity of treatment and a mortality of 33 per cent indicate a necessity for further study of this condition.

3. The patient is in double jeopardy from the obstruction and from the carcinoma. The obstruction must be treated first.

4. The age and the duration of the malignant disease add to the risk.

5. Obstruction of the colon is most often of the closed loop type.

6. In 31 cases the obstruction was in the left colon.

7. The clinical picture presents two phases: (1) longstanding signs and symptoms; (2) acute complete obstruction.

8. An exact preoperative diagnosis of the site of obstruction is important and can be readily determined by the flat x-ray plate of the abdomen and the barium enema.

9. Conservative treatment and resection (in the face of obstruction) cause a prohibitive mortality.

10. Simple loop colostomy of the right side of the transverse colon is the treatment of choice in obstructing carcinoma of the left colon.

11. Cecostomy, ileostomy, or ileotransverse colostomy are the procedures of

choice when the obstruction occurs in the right colon.

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EPIDEMIOLOGY AND RECENT DEVELOPMENTS IN POLIOMYELITIS*

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INTRODUCTION

Poliomyelitis as a disease has attracted the attention of lay individuals, laboratory workers and members of the medical profession for well over a century and a half.

The recognition of the existence of the disease is noted by many as dating back to 1600 B.C. Actually the first good clinical description of the disease is made by Underwood in a paper entitled "Debility of the Lower Extremities" published by J. Matthews in London, England in 1789.

In recent years a great deal of interest has been manifested in this disease and but few diseases can boast as much interest as has been centered around poliomyelitis. This interest has centered around the mode of transmission of the disease, prevention and treatment. A great deal of progress has been made along these particular fields, but the sum total of our knowledge of the disease is still rather limited—limited at least from the point of view of practical application.

ETIOLOGY

Although there are proponents of the bacterial etiology of the disease, it is generally recognized that the causative agent is a virus. There is also general agreement that the poliomyelitis virus is a neurotropic virus which probably is disseminated through the body by passage along the nerve fibers. More specifically, Toomey[1, 2] believes that the virus travels best along non-medullated or gray nerve fibers.

In 1909 Flexner and Lewis[3] described a virus as the etiologic agent of this disease and described in detail their experimental work with the M. Rhesus monkey. By cerebral inoculation of macerated spinal cord tissue, experimental animals developed a disease closely simulating infantile paralysis. The isolation of this virus and experimental work herein described have been repeated many times since.

THE PORTAL OF ENTRY

Although it is generally agreed that the virus of poliomyelitis is a neurotropic virus which enters the central nervous system by traveling along nerve tracts there has been no agreement as to ways and means by which this virus reaches the nerve tracts. There are two concepts which may be mentioned as to the portal of entry of the virus, namely the nasopharynx and the intestinal tract.

Toomey[1, 2] is the principal proponent of the gastrointestinal route of entry. By experimental inoculation of the virus into the gastrointestinal tract, Toomey was able to produce a disease in monkeys which simulated poliomyelitis. However, he was obliged to create severe stagnation of the gastro-

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intestinal tract before he could promote the development of this condition in his experimental animals. Other investigators are of the opinion that such unusual stagnation is probably non-existent normally and therefore the gastrointestinal work of Toomey and his associates should be looked upon as additional good scientific information without a great deal of practical application value.

Sabin and his associates[4] are proponents of the olfactory route of entry. This theory probably has more supporters than any other theory. Proponents of the theory point out that the virus has been isolated from the nasopharynx of cases and apparently healthy carriers; further that the epidemiologic characteristics of the disease suggest a droplet type of infection. Supporters of this theory also point out the ease with which entrance into the olfactory system might be brought about through the nasopharynx. Certainly the virus has been isolated in acute cases from the nasopharynx. All of these facts taken together are strongly suggestive of the nasopharyngeal route of entry of the virus.

ESCAPE

The escape and liberation of the virus from the human body have been repeatedly studied and innumerable publications are available for study.[5] The virus of poliomyelitis has been isolated from the stools and nasopharynx of apparently healthy carriers and persons acutely ill with the disease. The virus in the nasopharynx is usually present only for relatively short periods of time. The virus, however, may be present in the gastrointestinal tract weeks before the onset of clinical symptoms[6] and for months after the acute symptoms have subsided. There is of course a question which arises in the minds of investigators as to whether or not the primary localization of the virus is in the upper respiratory tract and that the gastrointestinal localization is secondary and is brought about by the swallowing of sputum contaminated with the virus.

RESERVOIRS

Although extensive research work has been directed at the recognition of reservoirs of this virus, there is but one definite reservoir known, namely, that of the human being. The reported recovery of the virus of poliomyelitis from a mouse found dead in a home where there had been a case of poliomyelitis only adds greater confusion to an already confused situation. To the best of the speaker's knowledge, this finding has not been repeated. Fowls, birds, rodents and various other animals have been examined but without satisfactory recognition of the poliomyelitis virus.

The virus has been isolated from sewage[7] and it has been shown that it will resist[8] the effect of chlorine in concentrations of 0.5 per 1,000,000 parts for over a half hour. There have been variously reported outbreaks which according to the epidemiological evidence would tend to incriminate food and milk, but all of these factors are affected by missing links of information and certainly it has been repeatedly pointed out that the pattern of behavior of poliomyelitis is such that it does not correspond with the characteristic behavior of food or water-borne infections.

The recognition of the virus in the nasopharyngeal secretions of persons affected with the disease and the recognition of the virus in sewage[7, 9, 10] and its presence in the stools of apparently healthy persons[5] lends emphasis to the significance of the human reservoir. The repeated recognition of the virus in apparently healthy human carriers and the rapid development of the carrier state is reported by Brown, Francis and Peerson. Certainly the report of the recognition of the virus of poliomyelitis nineteen days before the development of clinical symptoms in the stool of an apparently healthy individual[6] is a notable contribution to the knowledge of poliomyelitis.

MODE OF TRANSMISSION

There is no definite agreement as to the mode of transmission of this disease. The virus of poliomyelitis has been isolated from the nasopharyngeal washings of apparently healthy individuals as well as those suffering from the disease. It has been isolated from sewage, from food and

from the gastrointestinal tract of flies. This wealth of confusing facts only lends further confusion to the possible ways and means by which this disease is transmitted. It has also been proposed only because of some epidemiologic evidence that possibly the disease is transmitted by insect vectors.

Certainly the epidemiologic characteristics of the disease are such that they do not coincide with water or food-borne diseases. Water and food-borne epidemics are usually explosive in nature and affect large numbers of people simultaneously. Usually epidemics of poliomyelitis are progressive, develop gradually, show evidence of radial spread and reach their peaks within several weeks from the date of onset. The distribution of cases within the area affected is scattered with occasional aggregation, while in water-borne epidemics the distribution is somewhat more uniform. The attack of poliomyelitis in urban epidemics seldom exceeds one per one thousand and in rural epidemics the attack rates seldom exceed three per thousand. In food and water-borne outbreaks the attack rate among exposed persons is usually much higher.

One cannot too emphatically discount the food and water bases as the modes of transmission because as has been noted many times the rate of infection with poliomyelitis virus probably far exceeds the incidence of reported cases. Proponents of the food and water-borne mode of transmission therefore have this very important argument in their favor.

Probably the oldest and the best theory of the mode of transmission of poliomyelitis is that of person to person contact and droplet infection. Certainly the number of persons succumbing to the disease who have had contact with other cases is statistically significant. An investigation which has been carried on evidence of contact with cases has been reported as high as 60 to 80 per cent. There are contradictory points to this method of transmission. The low secondary attack rate, intra and extra-familially, is certainly suggestive of some important extrinsic or intrinsic factors limiting the occurrence of secondary cases. There is also the important fact that additional cases in families and households may develop in subsequent years in persons who escaped the first exposure.

AGE, SEX AND RACE DISTRIBUTION

The disease affects primarily persons between the ages of five and fourteen years of age with a reasonably high incidence under four and with greatest concentration of cases between five and nine years. The disease, however, does affect persons of all ages and the speaker has had the experience of seeing a case seventy years of age and has seen poliomyelitis in a new-born infant.

By sex, the distribution of the disease shows a somewhat greater incidence among males than among females, the ratio being approximately 1.4 males to one female. A great deal has been said and written about the racial distribution of poliomyelitis depending almost entirely upon the section of the community or section of the country which is affected. My associates and I have not been able to show any difference in the race distribution of poliomyelitis. By individual epidemics difference of significant amount has not been noted, but over a period of many years the attack rate among negroes and whites is approximately the same. The differences which have been noted in attack rate by race were due to geographic or area location of the epidemic.

The rural as against the urban incidence of the disease is noteworthy. In the most severe urban outbreaks the incidence of the disease seldom exceeds one per 1,000 population while in rural areas when epidemics occur the attack rates are two or three times as great as in urban areas. As a matter of fact the actual incidence of the disease—the rural against urban—shows a much greater incidence in the rural areas than in the urban. The rural incidence of poliomyelitis is so much greater that it is frequently referred to as primarily a rural disease. The Henderson County outbreak of 1945 in Tennessee is a good illustration of this theory.

There is a definite seasonal variation of the disease. It has been repeatedly noted

that poliomyelitis has an apparent selective occurrence both as to season and individuals. Epidemics seem to occur in summer and early fall and as previously noted, clinical poliomyelitis affects but a very few persons who live in the epidemic area.

SEASONAL VARIATION

The seasonal variation is of significant epidemiologic importance because coupled with this seasonal variation is the fact that the incidence of paralytic disease in areas where seasonal changes are not so radical is considerably lower than in the more temperate climates where the seasons change and change radically. This has brought about the prognostication that possibly there is something in the physiology of the human being which in part at least is related to the possibility of successful and clinical invasion of the virus. In other words, the prognostication is that a certain percentage of the people in these areas fail to make adequate physiologic adjustments with the varying climate and season with the end result that the infection with poliomyelitis virus results in clinical disease. On the other hand, the person whose physiology follows a more favorable pattern of adjustment is infected but escapes clinical disease.

PREDISPOSING FACTORS

There is a great deal of evidence to suggest that there is a hereditary predisposition to poliomyelitis and that the clinical disease is prone to recur in families. This fact is proposed along with drawing attention to the fact that the incidence of secondary cases in households during the same epidemic is rather infrequent. In a survey of the incidence of poliomyelitis in families, Aycock pointed out that 51 per cent of the cases gave a history of disease among relatives while only five per cent of his controls gave a similar history.[11, 12]

Further investigations by other workers, notably Adair and his associates[13] confirm this familiar predisposition to poliomyelitis. There have been attempts by Aycock and Draper to associate the disease with endocrine imbalance and to point out that certain institutional types[14] are more apt to acquire clinical poliomyelitis than are others. Aycock further suggests that susceptibility may reside in a subclinical endocrine difference and more particularly a subclinical difference between periods of growth and development. These theories needless to say cannot be completely ignored but evidence to the contrary is proposed by other investigators.

Aycock also points out that there appears to be a predisposition to attacks of poliomyelitis among pregnant women. He noted that poliomyelitis is associated with pregnancy about four times as frequently as it would be expected in non-pregnant individuals. The closer examination of this relationship shows that the greatest risk occurs in the second and third trimester of pregnancy.[15]

Here again Aycock draws attention to the fact that this may be associated with some endocrine disturbance. Experiments have been attempted by Jungblut to associate the incidence of the disease with vitamin C deficiency. Helm[16] has proposed that vitamin B is the major factor for increased susceptibility for the virus and Weaver in turn[17] in his experimentation with cotton rats was unable to show any relationship between vitamin B deficiency and the poliomyelitis virus.

It is conceivable therefore that the endocrine and vitamins, or the lack of endocrines or vitamins, is associated with susceptibility to clinical poliomyelitis. If there is an intimate association the method of its operation is not clearly understood.

Certainly from the overwhelming amount of information which is available, it is obvious that the incidence of infection with poliomyelitis virus far exceeds the reported incidence of clinical disease.

It has also been noted by some investigators that trauma, over-exertion and exhaustion are predisposing to clinical poliomyelitis. One must question this proposition because although trauma may be very definite, the degree of trauma may be variable or at least the interpretation placed upon the severity of such trauma by the investigator is variable. Also, exhaustion and overexertion are relative terms. In the Detroit

studies virtually no correlation existed between these factors and the incidence of the disease. The incidence of severe poliomyelitis among recently tonsillectomized persons is quite significant. For example, there is evidence to indicate that recently tonsillectomized children develop bulbar poliomyelitis much more frequently than those children who have not recently been tonsillectomized.[18] There are, of course, persons who do not agree with this. But the evidence is overwhelmingly in favor of the proponents of the predisposing effect of tonsillectomies to the development of bulbar poliomyelitis.[19]

CARRIERS

As has been previously noted the incidence of infection with poliomyelitis virus far exceeds the incidence of clinical cases.[5, 6] My associates and I were able to show that in one outbreak involving an institution caring for children between infancy and ten years of age, several individuals without any clinical symptoms were found to be carriers of the virus of poliomyelitis. A nurse and a physician, in addition to several of the children, who had intimate contact with cases were found to be positive. It seems quite possible that abortive and subclinical cases may carry the virus for varying periods of time. Brown and his associates, for example, were able to show that a person who developed clinical poliomyelitis had actually harbored the virus of poliomyelitis in his stools nineteen days before the onset of the first clinical symptoms.

PREVENTION

The principals of prevention of poliomyelitis have varied many times even within the past decade. Vaccines have been developed which have been most unsuccessful. Prophylactic spraying of the nasopharynx with various and sundry chemicals has proved to be a total failure. As a matter of fact there are observers who believe that both the vaccine and the nasal spraying had actually the opposite effect of that which was expected.

The closing of schools, the avoidance of crowds and the isolation of the patient and contacts appear to have the effect of only a placebo. Certainly the wide and general distribution of the virus in nature and among humans particularly precludes the possibility of any effective means of avoiding possible infection. Based upon these facts the rules and regulations for the control of communicable diseases have brought about a change in attitude towards the isolation of poliomyelitis cases and contacts. Presently but few states isolate cases and contacts.

SUMMARY

The only significant statement which a person can make as far as prophylaxis of poliomyelitis is concerned is that up to this time nothing has been found to be effective and the only encouraging statement which can be made is that clinical and research efforts are ever expanding in the hope of finding some effective ways and means through which preventive measures may be exercised.

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DICOUMARIN—MEDICAL ASPECTS*

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NEW ORLEANS

To the great discredit of the internist, thrombo-embolism is today widely regarded as a surgical problem. Actually, of course, it is a phenomenon apt to arise whenever bed rest and immobilization for even a few days are imposed upon the patient for any reason, but the medical profession is indebted chiefly to the surgeon for the current interest in this insidious threat to life. There is as yet no unanimity of opinion as to the optimum methods of prevention and treatment but new weapons are available and new records are being set. This paper will be concerned with the application of one of the newer anticoagulants, dicoumarin, [3,3′-methylenebis (4-hydroxycoumarin)], to the prevention and treatment of intravenous clotting.

THROMBO-EMBOLISM OF PERIPHERAL ORIGIN

Those who feel that venous thrombosis is predominantly a postoperative problem should examine the careful leg dissections reported by Hunter and coworkers[1] which showed that intravascular clots developed in about 60 per cent of middle-aged and elderly individuals put to bed for even a few days, an event generally attributed to circulatory stasis and possibly also to hypercoagulability of the blood. Similar studies have, of course, been made before, and all authors are agreed on the frequency and menace of these loosely attached clots.

There is considerably less agreement, however, regarding the best method of coping with venous thrombosis. The prophylactic importance of ambulation or leg exercises is illustrated by the observation that the incidence of venous thrombosis in the legs of those so treated up to 48 hours before death was 18 per cent, whereas in those inactive prior to death it was 53 per cent.[1] The further importance of deep breathing to accelerate the return of blood to the thorax and of compression bandages around the leg to increase the rate of blood flow through the deeper veins has been repeatedly advocated by Ochsner.[2] No one can estimate the number of lives which have been saved by the adoption of these sensible measures, but certainly no one can maintain that they are completely effective. We have, for example, in one year seen seven deaths from pulmonary embolism on a hospital service where these measures have been routinely adopted and keenly enforced.

Because prophylaxis against clotting frequently fails, venous ligation is now widely advocated as a method of protecting the patient from detachment of the clot. Bilateral ligation of the femoral veins is said to be a safe and adequate method of preventing thrombosis and embolism[3] but the reasons for this confidence are not entirely clear. It probably is a satisfactory way of dealing with thromboses limited to the lower legs[4] but one is entitled to suspect that the surgeon can seldom be sure that the ligatures are always placed proximal to the danger zone,[5] and there is some reason to believe that the ligature itself induces thrombosis central to it.[6] Pulmonary embolism has occurred in 5 per cent of cases following bilateral ligation of the superficial femoral veins[7] and in an effort to control detachment of clots from the vena profunda femoris, ligation of the common iliac vein has been advised.[8] It is not within the province of this paper to evaluate the relative merits of the various operations but the principle involved seems to lead to the conclusion that the only vessel which can be ligated with confidence is the inferior vena cava.[9] This seems too radical a procedure except in cases of suppurative pelvic thrombophlebitis.[10]

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The staunchest advocate of venous ligation[3] writes that interruption of the superficial femoral vein is an effective method of treatment but that "Patients with continued infarcts following this procedure should have anticoagulant therapy. Those with continued pain and swelling . . . should be treated by lumbar sympathetic procaine blocks." These qualifications seem to bespeak a lack of total confidence in the surgical treatment and to justify consideration of certain chemical methods now available.

Objection to the use of anticoagulants has been voiced by DeBakey,[11] and it is certainly hazardous therapeutics to treat one disease by substituting another. A very large number of patients has been treated with consistently impressive results, however. No discussion of the many favorable experiences with heparin can be given here.[12, 15] Barker and associates,[16] who gave dicoumarin to 1000 patients postoperatively, reported one embolic death and that was in a patient whose prothrombin time had been allowed to return to normal. Barker[17] gave the drug to 180 patients with pulmonary embolization; only 1.1 per cent of these had subsequent infarction with a 0.6 per cent mortality rate whereas a control group of 678 patients exhibited a 43.8 per cent incidence of recurrence and a mortality rate of 18.3 per cent. Levan[18] noted recurrence of slight superficial thrombophlebitis in only one of 60 cases. Murray[6] found no instance of thrombo-embolism in 400 "dicoumarinized" postoperative patients; 149 patients with pulmonary embolism were treated without a death and 371 cases of peripheral venous thrombosis were managed without pulmonary embolization and with a lower incidence of postphlebitic edema and varicosities. Under conservative treatment Jorpes[15] had a 16 per cent mortality rate in 543 cases of disease of the peripheral vein but anticoagulant therapy (heparin and dicoumarin) applied to 900 patients reduced the mortality rate to 0.67 per cent. In a summary of a very large experience with heparin in Sweden (there is no reason to think that essentially similar results might not be obtained with dicoumarin) Zilliacus[14] demonstrated a mortality rate of less than one-tenth of that in conservatively treated cases; 21 of 65 patients with pulmonary embolism treated non-specifically died, but there were no deaths in 103 heparinized patients. Eruzelius[19] reduced the mortality rate of thrombo-embolism to one-third by using dicoumarin and the postoperative death rate of pulmonary embolism from 0.49 per cent to 0.16 per cent. Allen,[20] who gave dicoumarin to 1628 surgical patients, saw a dramatic reduction in the incidence of thrombo-embolism.

This list is by no means complete but it shows what can be achieved. It no longer seems necessary to choose *between* venous ligation and anticoagulant therapy; one supplements the other and judicious combination of the two should provide almost complete protection. Venous ligation appears to be unnecessary in cases of acute non-suppurative thrombophlebitis; dicoumarin gives far better results and lumbar sympathetic block with procaine hydrochloride may also be profitably employed if vasospasm co-exists. Obviously, however, blocks cannot be repeated during administration of dicoumarin for fear of hemorrhage. The patient with phlebothrombosis deserves the double protection offered by venous ligation and anticoagulants. Although it obviously is neither feasible nor necessary to give dicoumarin to every patient who takes to bed because of illness, its prophylactic use in persons over 40 years of age who face a period of immobilization and who present no contraindications should be seriously considered. Allen, Linton and Donaldson[21] noted that a roughly similar plan resulted in the "dicoumarinization" of 16.5 per cent of 985 surgical patients and a lowering of the incidence of thrombosis and embolism to about 25 per cent of a control series.

THROMBO-EMBOLISM OF CARDIAC ORIGIN

Coronary thrombosis: It has long been known that the chambers and valves of the heart are occasionally a source of emboli but the frequency of this event is not as

widely appreciated as it should be. The incidence of mural thrombi in myocardial infarction is especially high, the estimates ranging from 17 to 83 per cent.[22, 32] The frequency with which portions of these thrombi detach themselves and cause clinically detectable disturbances in either side of the circulation is difficult to estimate. Blumer[27] reported that 50 per cent of his patients had mural thrombi and that in 14 per cent of these emboli were clinically detectable. Bean[29] noted that 75 per cent of patients with mural thrombosis of the right ventricle had pulmonary embolism but that fatalities were due to large emboli arising from clots in the pelvis and legs. Nay and Barnes[31] estimate that the incidence of pulmonary embolism in clinical series of coronary occlusion is 3 per cent and, in postmortem series, 42 per cent. Pulmonary embolism accounts for from 5 to 11 per cent of the deaths which occur in the convalescent period.[28, 29, 33]

Final decision regarding the efficacy of anticoagulants in the prevention and treatment of this form of thrombo-embolism must be postponed, but encouraging experiences have been reported. Although dicoumarin might be expected to prevent extension of the original coronary thrombus or recurrence elsewhere in the coronary tree, its chief usefulness is in the prevention of mural and peripheral venous thrombosis. Solandt and Best[34] reported that heparin could prevent mural thrombosis experimentally but dicoumarin is the drug of clinical choice since Mallory[35] showed that mural thrombi do not form until the sixth postinfarction day and that such clots are organized probably by the sixteenth day. An additional argument for its use rests upon the claim that there is a tendency toward hypercoagulability of the blood in coronary occlusion [36, 38] and during the administration of digitalis[39, 43] and xanthine derivatives.[44] The changes are at best not striking, and large doses of aminophylline given to man have produced no significant alterations in the prothrombin time.[45] The effect of digitalis on the prothrombin time has not been confirmed.[46]

No attempt has been made to assemble all the cases of patients with myocardial infarction who have been treated with anticoagulants. The first report was that of Nichol and Page,[32] who treated 50 attacks in 44 patients with an immediate mortality rate of 16 per cent but in their six autopsies they noted no mural thrombi or emboli. Peters, Guyther and Brambel[38] reported a series of 50 "dicoumarinized" patients with an immediate mortality rate of 4 per cent and a 2 per cent incidence of clinical embolism as opposed to a control group of 60 cases in which the mortality was 20 per cent and the incidence of embolism 16 per cent. Wright[47] reported favorable experience with 76 patients but gave no figures. Parker and Barker[48] used dicoumarin alone or in combination with heparin in the management of 50 patients with acute myocardial infarction; the mortality rate was 10 per cent and the incidence of thrombo-embolism was only 4 per cent.

Auricular fibrillation: The occurrence of embolism from detached auricular thrombi is unpredictable[35] and no evidence proving that anticoagulants will diminish the hazards has been published.

Congestive heart failure: No studies on the effect of anticoagulants upon the incidence of thrombo-embolism in congestive heart-failure not due to myocardial infarction have appeared but there is no apparent contraindication to their use and every likelihood that good results will be achieved.

Bacterial endocarditis: It is now thought unwise to use anticoagulants in this condition because of the accompanying capillary fragility and the tendency to spontaneous hemorrhage.

MISCELLANEOUS CONDITIONS

Cerebrovascular disease: Young[49] has advocated dicoumarin in the management of patients with hypertensive encephalopathy, cerebral arteriosclerosis and cerebral thrombosis but this appears to be dangerous. Dicoumarin should always be used cautiously in patients with hypertension and it is notoriously difficult to differentiate clinically between small hemorrhages and thromboses in the brain. The drug is probably useful in the treatment of thrombosis

of the larger intracranial veins, however. Its application to multiple sclerosis does not look promising.[50, 51]

Hypercoagulability of the blood: Dicoumarin might be useful in controlling the tendency to thrombocytosis which commonly occurs in patients with leukemia, Hodgkin's disease and polycythemia vera, and following splenectomy but no studies of statistical value are available. Carcinoma of the body of the pancreas is another condition frequently associated with peripheral thromboses. Preliminary studies are also underway concerning the usefulness of anticoagulants in the management of thromboangiitis obliterans, arteriosclerosis obliterans, frost-bite, trench foot and arterioembolism of the extremities.

DOSAGE AND ADMINISTRATION

There is no standard schedule for administration of dicoumarin since patients vary in body weight and in sensitivity to the drug. Enough must be given to maintain the concentration of prothrombin in the blood between 10 and 30 per cent of normal.[21] Daily estimations of prothrombin time by a reliable method are imperative, and treatment must not be started until this value is known. When normal, the initial dose is 300 mg. by mouth and 200 mg. is given on the following day. No more is given until the prothrombin time for the day is known. On each subsequent day that the plasma prothrombin concentration is 20 per cent or more, a single dose of 200 mg. is given but none is given if it is less than 20 per cent. The maintenance dose may vary from 100 to 300 mg., however, since individuals may differ in sensitivity. The drug has a delayed action of from 24 to 48 hours and hyperprothrombinemia may persist for many days after its use has been discontinued. Patients with cardiac disease are said to be hypersensitive and smaller doses may be required.[16] The strictest laboratory control is essential if hemorrhage is to be prevented.

COMPLICATIONS

The only important danger from use of anticoagulants is hemorrhage. Aggeler[52] collected 1471 cases in which hemorrhagic death due to dicoumarin occurred in 0.34 per cent and less important hemorrhage in 8.3 per cent; the latter figure, however, included many cases of microscopic hematuria, which is not in itself an indication to discontinue using the drug and also cases in which the prothrombin time had been allowed to drop below 10 per cent. Of the 50 patients treated by Peters, Guyther and Brambel[38] the only evidence of bleeding was microscopic hematuria in three. Evans[53] reported two hemorrhage deaths in 56 patients and an incidence of spontaneous bleeding in 14 per cent. Bruzelius[19] found that 4.8 per cent of 1656 patients bled but only half of them importantly, and there were two deaths in this large series. Barker and associates[16] saw major bleeding in 2.5 per cent of 1000 patients, minor bleeding in 3.9 per cent. Gefter, Kramer and Reinhold[54] had eight cases of mild hemorrhage and no deaths in 30 cases. Minor hemorrhage (epistaxis, hematuria, petechiae and ecchymoses) occurred in 3.1 per cent and major hemorrhage in 1.9 per cent of Allen's large series.[20] In the series of 50 cases reported by Parker and Barker[48] the prothrombin concentration was difficult to control in 36 per cent of the patients and three of them exhibited minor hemorrhages. Bleeding due to dicoumarin may be controlled by the transfusion of blood and the intravenous administration of menadione bisulfite in doses of 50 to 100 mg. every four hours until the prothrombin concentration of plasma is restored to 30 per cent or more. All potent drugs are dangerous in inexperienced or careless hands and dicoumarin is certainly no exception, but its controlled use is perfectly justifiable.

CONTRAINDICATIONS

There are certain conditions which definitely preclude full dosage schedules of dicoumarin: (1) Blood dyscrasias accompanied by purpura are obvious contraindications; (2) hepatic disease must be approached cautiously if it is accompanied by prothrombin deficiency; (3) renal insufficiency of any degree means that the elimination of dicoumarin will be retarded and its effect therefore cumulative; (4) subacute bacterial endocarditis is not a suitable disease for anticoagulant therapy since

spontaneous hemorrhages are a common feature; (5) ulcerative lesions and recent operative wounds are apt to bleed; (6) patients who are being treated with drainage tubes at various sites are likely to bleed at points of mechanical pressure; (7) recent operations on the brain or spinal cord must be regarded with caution because minute areas of hemorrhages are apt to produce serious disturbances; (8) it would appear unwise to give the drug to pregnant or parturient women.

CONCLUSIONS

Dicoumarin is an effective but potentially dangerous agent in the prevention of intravascular thrombosis and embolism. It is useful to the surgeon as an adjunct in the prophylaxis and treatment of thrombophlebitis and phlebothrombosis. It should be employed by the internist whenever the tendency to thrombosis is increased because of enforced bed rest, general debility, certain blood dyscrasias or heart disease.

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A REVIEW OF CERTAIN ASPECTS OF THE PATHOLOGIC PHYSIOLOGY OF HEART DISEASE

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NEW ORLEANS

In view of the greatly renewed interest in congenital heart disease and in the mechanism of congestive heart failure, it is felt that a review of some features of the abnormal physiology involved in these conditions would be timely. Of especial interest are the data collected from direct catheterizations of the right side of the heart. The investigators in this field have contributed immeasurably to a more lucid understanding of the hemodynamic conditions present in various congenital anomalies and in congestive heart failure. The four congenital diseases of the heart (only ones discussed) in which most of the observations have been made are: (1) patent ductus arteriosus; (2) interauricular septal defect; (3) interventricular septal defect, and (4) the tetralogy of Fallot.

Of considerable importance are the changes occurring in the lungs and pulmonary circulation. These abnormalities in pulmonary dynamics are manifested mainly by: (1) alterations in the blood pressure in the pulmonary artery, veins, and capillaries; (2) chronic pulmonary congestion; (3) acute pulmonary edema; (4) cardiac dypnea; (5) alterations in vital capacity, and (6) alterations in intrapleural pressure.

CONGENITAL HEART DISEASE

Patent ductus arteriosus: Normally, at birth, every infant possesses a patent ductus arteriosus, but in most instances the lumen gradually is occluded, so that by the end of the second or third month the only remaining vestige is a thin fibrous cord which, from that time on, serves no useful function. Not infrequently, however, the ductus fails to involute and persists as a short patent tube between the descending aorta and the pulmonary artery; this anatomic abnormality has deleterious physiologic effects on both the pulmonic and systemic hemodynamics. When a patent ductus arteriosus obtains, there is a marked difference in the output of each ventricle: the right ventricle receives blood only from the right auricle, which has previously received the same from the systemic circulation via the superior and inferior venae cavae: the right ventricle ejects its column of blood into the pulmonary artery, which already is partially filled with blood entering it from the patent ductus; after traversing the pulmonary circuit, the blood is received by the left ventricle from the left auricle and ejected into the ascending aorta, only to have a part of it "drained off" by the patent ductus and shunted into the pulmonary artery; thus, the output of the left

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ventricle amounts to that quantity put out by the right ventricle plus the additional amount carried via the patent ductus arteriosus.

In order to obtain more definite information regarding the circulatory changes in the pulmonic and systemic circulation resulting from this congenital anomaly, Eppinger, Burwell, and Gross[1] created an artificial ductus in each of five dogs by the surgical anastomosis of the left subclavian artery to the main pulmonary artery. This afforded them opportunity to procure samples of blood: (1) that came directly from the right ventricle; (2) that went into and out of the lungs, and (3) that was ejected into the aorta from the left ventricle. Data received were applied to the Fick principle so as to determine the output of each ventricle. It was calculated that in each of the five dogs the amount of blood shunted back into the pulmonary circulation from the aorta via the ductus was more than 50 per cent of the total quantity of blood put out by the left ventricle; it is obvious at once that the systolic outflow of the dogs' left ventricles was over twice that of their right. To illustrate, data from one of their dogs are as follows:

Output of each ventricle in intact animal—2.05 liters per minute.

Output of left ventricle with shunt—2.83 liters per minute.

Output of right ventricle with shunt—1.36 liters per minute.

Volume shunted from aorta to pulmonary artery—2.83 minus 1.36 = 1.47 liters per minute.

$$\text{Per cent of output of left ventricle shunted} = \frac{1.47 \times 100}{2.83} = 51.9 \text{ per cent.}$$

Then too, it was found by chemical analysis that the mixed blood (from both right ventricle and ductus) going into the lungs was significantly higher in oxygen content than that coming alone from the right ventricle; this substantiated the previously held assumption that a patent ductus resulted in an arteriovenous shunt, with the direction of flow from aorta to pulmonary artery. Furthermore, by use of water manometers, the authors measured the pulmonary arterial pressures in three of their five dogs, both before and after creation of the artificial ductus; they observed that in two of the dogs the pressures were significantly elevated following the anastomoses. Measurements of blood volumes in the operated dogs revealed increases in two of them; in one instance the blood volume before was 1975 c.c., and 3880 c.c. subsequent to operation.

In addition to their work with experimental animals, Eppinger et al. collected data on six patients, each with a patent ductus arteriosus uncomplicated by other congenital defects; each patient underwent surgical ligation of the ductus. Using methods comparable to those above, they found that the amount of blood shunted through each ductus varied from 45 to 77 per cent of the quantity ejected with ventricular systole; therefore, the left ventricular output was varied directly with the diameter of the ductus, the smallest of which was 7 mm. Other observations of interest included: (1) low diastolic and unusually high pulse pressures in all patients; (2) increased circulation time and total blood volume in each of two patients, and (3) no significant variations in venous pressures or vital capacities.

It can be seen, therefore, that the left side of the heart suffers most of the indignities imposed by a patent ductus arteriosus, the increased amount of work it must perform being proportional, roughly, to the size of the arteriovenous shunt. But the right ventricle is not immune altogether: during ventricular diastole, when the pulmonary valves are closed, blood is pouring into the pulmonary artery from the patent ductus (assumption based upon the diastolic component of the continuous murmur heard in most cases of patent ductus); this may result in an elevation of pulmonary arterial pressure, and during the next systole the right ventricle has more difficulty in opening the pulmonary valves. But this bit of additional work which may result is in no way comparable to that done by the left side of the heart, and this is borne out clinically,

since, as determined by roentgenography and fluoroscopy, left-sided enlargement usually exceeds right-sided enlargement.

Morphologic changes also occur in the pulmonary vessels. One frequently can detect, by fluoroscopy, an increase in the size of the main pulmonary artery and/or its large branches, due possibly, to three factors: (1) chronic elevation of intrapulmonary arterial pressure secondary to an almost constant inflow of blood from the aorta via the patent ductus; (2) continuous bombardment of the walls of the pulmonary artery by a stream of blood from the ductus, and (3) chronic intrapulmonary capillary hypertension secondary to increased venous pressure following left-sided failure, if it can be assumed that left-sided failure of necessity results in increased venous pressure. Due to the abnormally large volume of blood transmitted by the main branches of the pulmonary artery, increased pulsations of these branches upon fluoroscopic examination are often noted and, in addition, a so-called "hilar dance," seen as a pulsating systolic dilatation of the smaller vessels in the root of each lung.

Bloomfield et al.[2] were the first to study directly, by use of the intracardiac catheter and a Hamilton manometer, the right heart pressure in normal subjects. They found that the mean right auricular pressure, with respect to atmospheric pressure, varied from minus 2 to plus 2 mm. mercury, that the maximum systolic pressure in the right ventricle varied from plus 18 to plus 30 mm. mercury, with an average of 25 mm. mercury, and the diastolic from minus 7 to plus 2 mm. mercury. Recently, Hickam and Cargill,[3] by means of intracardiac catheterization, made manometric determinations of pulmonary arterial pressures in normal subjects before and after moderate exercise, and found no significant changes in pressures as a result of exercise; they concluded that the pulmonary circulation can accommodate efficiently to moderate increases in blood flow without increasing the mean pressure. Dexter and Burwell[4] report a case of patent ductus arteriosus in a three-year old female child in whom the right ventricular pressure was 54/1 mm. mercury, and the pulmonary arterial pressure was 55/39 mm. mercury. As far as I have been able to determine, this is the only case of patent ductus so far reported in the literature wherein intracardiac catheterization was made to accurately determine the pressures. Cournand[1] states that the average normal systolic pressure in the pulmonary artery is 25 mm. mercury (identical with the average normal systolic pressure in right ventricle), and the average normal diastolic pressure 8 mm. mercury. It can be seen, then, that in the case cited above the patent ductus led to a marked increase in both the systolic and diastolic pressures in the pulmonary artery.

Interauricular septal defect: In most cases where there exists an abnormal communication between the left and right auricle, the opening is quite large and allows a considerable quantity of blood to be shunted from left to right—an arteriovenous shunt; this usually results in tremendous enlargement of the right auricle, right ventricle, and pulmonary arterial tree, since these structures are handling a greatly increased volume of blood. Whereas, it had been a clinical assumption that the flow of blood was from left to right, Brannon et al.[5] proved that such is the case in interauricular septal defects after studying four patients, in each of whom the diagnosis was made clinically. They found that the oxygen content of the right auricular blood was far in excess of that found in blood from both venae cavae, thereby indicating that oxygenated blood was actually flowing from the left to the right auricle. The amount of blood passing through the shunt varied from 52 to 71 per cent of that quantity received from the lungs by the left auricle, and in all cases the output of the right ventricle was over twice that of the left ventricle. In two cases the systolic right ventricular pressure was determined and found to be 40 and 41 mm. mercury. Richards[6] reports a case of interauricular septal defect in which the right ventricular pressure was 59/6 mm. mercury, and the pulmonary ar-

terial pressure 33/10 mm. mercury, the former is over twice the normal, while the latter is only slightly above the average normal value.

Interventricular septal defect: Whenever there is a defect in the interventricular septum, the output of the right ventricle is more than that of the left, if the theory of a left to right shunt is correct; it not only ejects all the blood returning from the systemic venous circulation delivered to it by the left ventricle, but also that amount forced into it from the left ventricle via the defect. Baldwin et al.[7] did intracardiac catheterizations on two patients with interventricular septal defects and found that the oxygen content of the blood in the right ventricle was significantly higher than that of blood in the right auricle in each case, indicating a flow of oxygenated blood from left to right. In one case, the right ventricular systolic pressure varied from 33 to 43 mm. mercury, the diastolic from 16 to 10 mm. mercury; in the other case, the right ventricular systolic pressure varied from 54 to 47 mm. mercury, the diastolic from 25 to 20 mm. mercury, thus exceeding the average normal value of 25/2 mm. mercury. The pulmonary arterial pressures were not measured, but it seems logical to think that they too would have been abnormally elevated, for Richards[6] cites a patient with an interventricular septal defect in whom the right ventricular pressure was 74/7 mm. mercury, and the pulmonary arterial pressure 74/25.

Tetralogy of Fallot: In this congenital anomaly there is a stenotic pulmonary artery, an interventricular septal defect, and a dextraposed aorta overriding both the left and right ventricle; this set of anatomic changes places a strain upon the right ventricle leading to its enlargement; it is the most common congenital cardiac defect producing cyanosis in adults and children over one year of age.[8] Since a large quantity of the venous blood that is ejected by the right ventricle and passes directly into the overriding aorta without going to the lungs to be oxygenated, one would not expect the pressure in the diminutive pulmonary artery to be elevated, but to be either normal or decreased. Richards[6] reported observations made in a case of tetralogy of Fallot: the pressure in the "free" portion of the right ventricle was 98/1 mm. mercury, while that in the underdeveloped infundibular portion was only 13/4 mm. mercury, upon passing the catheter into the stenosed pulmonary artery the pressure was found to be 12/8 mm. mercury, and in the left branch 18/10 mm. mercury.

COMPENSATED ACQUIRED HEART DISEASE

Even before frank congestive left heart failure occurs in patients with valvular heart disease, or yet prior to the appearance of any clinical symptoms which might make one suspect incipient failure, certain changes in cardiac function may be brought forth by special procedures. Stewart et al.[9] studied 40 patients with various types of the common valvular disorders not in failure along with 13 normal subjects as controls. They found an average decrease in both the cardiac index (cardiac output per square meter of body surface) and in the average stroke volume per kilogram in those patients with valvular heart disease as compared to the controls. This might infer that already increased pulmonary resistance, secondary to early pulmonary capillary congestion, was present, thereby causing a decrease in the amount of blood flow to the left ventricle. In fact, they found a slight increase in the arm to tongue circulation time in the majority of their cardiacs. Bloomfield et al.[2] studied a case of rheumatic heart disease with mitral insufficiency and stenosis, and aortic and tricuspid insufficiency, both during failure and after compensation had been established. By right heart catheterization, they noted that even after failure was overcome clinically, the right ventricular systolic pressure remained abnormally high—57 mm. mercury; normal 25 mm. mercury. They feel that this persistent elevation of pressure may result from continued inefficiency of the left auricle with subsequent pulmonary vascular hypertension, plus the effects of arterial thickening, arteriolar sclerosis and necrosis, and increase in collagen through-

out the interstitial tissue of the lung, which Parker and Weiss[10] believe to be characteristic pulmonary changes in mitral stenosis. Bloomfield et al.[2] noted that no abnormality in pulmonary dynamics occurred in patients with uncomplicated arterial hypertension who did not exhibit clinical congestive heart failure.

CONGESTIVE HEART FAILURE

The alterations in pulmonary dynamics occurring with, or as a result of, the clinical syndrome of congestive heart failure are primarily dependent upon congestion in the pulmonary capillaries. It is pertinent, therefore, to discuss here the anatomic changes in the lung parenchyma associated with, and the physiologic effects of, pulmonary congestion.

Anatomic changes in the lung parenchyma: The degree of pulmonary congestion roughly parallels the severity of the cardiac failure as judged by the physical signs and symptoms accompanying the condition. In the earliest stages of left congestive failure there may be only dilatation and engorgement of the lung capillaries without clinically detectable edema. Lambert and Gremels,[11] by electrical methods, can detect the occurrence of this pre-edematous stage. If the pulmonary congestion progresses, pericapillary edema ensues, and enough fluid leaks into the alveolar spaces to give rise to clinically apparent fine, moist rales. If a patient develops congestive heart failure severe enough to cause death, then at the autopsy table marked changes in the lung will be seen. Harrison[13] states unequivocally that all such patients invariably exhibit congestion and engorgement of the lungs; such lungs are enlarged, swollen, and heavy with edema fluid, and upon section exude copious amounts of frothy fluid which may or may not be grossly blood-tinged. Upon microscopic examination, one encounters marked capillary dilatation and engorgement, edema of the interstitial spaces, and alveoli which are partially or completely filled by the transudate from the capillaries; the alveoli may show albuminoid casts[14] and large, pigment-containing "heart-failure cells," in addition to a varying number of red blood cells.

Physiologic effects of pulmonary congestion: Once the lungs of a patient in congestive heart failure become the seat of pulmonary congestion, dyspnea ensues, the vital capacity decreases, the normally moderately negative intrapleural pressure becomes more negative, the velocity of the blood flow decreases, and the pulmonary arterial pressure rises.

Dyspnea is often the first clinical evidence of left congestive heart failure, and is frequently the patient's only or major complaint. Just what is responsible for this distressing symptom, and by what mechanism does it appear? By definition, dyspnea is any breathing associated with undue effort whether consciously or subconsciously[15]; only those forms of dyspnea resulting from heart disease — cardiac dyspnea—will be detailed.

In past years it was thought and taught that cardiac dyspnea resulted from the effects of decreased oxygen content and increased hydrogen ion concentration of the blood in contact with the respiratory center, these alterations in the circulating blood supposedly resulting from the inability of the pulmonary capillaries to take on oxygen from, and give up carbon dioxide to, the alveolar air in the presence of an intervening wall of edema fluid. Since this popular and highly attractive theory could not be supported by one shred of concrete evidence,[15, 16] it wavered on its shaky foundations and tumbled into oblivion.

Then how is cardiac dyspnea produced? Legallois,[17] in 1812, first demonstrated that respirations are slowed and increased in depth when the vagus nerves are severed, then Hering and Breuer[18] showed that vagal section inhibited expiratory chest movements normally initiated by the inspiration of air. The Hering-Breuer reflex is of paramount importance in the nervous control of ordinary respiration. Christie[15] states, "There are certain nerve endings in the lungs which act as stretch receptors, and when the lungs are inflated by inspiration, these stretch receptors discharge impulses

which travel to the respiratory center via the vagus nerves which inhibit inspiration, and, at the same time, increase the rate of respiration." That pulmonary congestion is a direct cause of dyspnea has been proven by numerous investigators.[15] As seen above, the lungs in pulmonary congestion are big and bulky, stiff, taut, and rigid; their alveolar walls are stretched, their air sacs distended with fluid. This marked increase in tension of the lung tissue makes the Hering-Breuer reflex hyperactive; that is, when only a small amount of air is drawn into the lungs, premature impulses pass up the vagi to the respiratory center and cut short the inspiration, and, as a result of this premature initiation of the Hering-Breuer reflex, the respirations are shallow and rapid. To prove that cardiac dyspnea is mainly reflex in origin, many workers, among whom are Harrison et al.[19] have produced experimental pulmonary congestion in animals and noted that the resulting dyspnea is abolished by cutting of the vagus nerves.

Although pulmonary congestion is considered to be the major cause of cardiac dyspnea, there is some evidence that other factors may play contributing roles. For instance, Mills, cited by Hoff,[20] produced transient hyperpnea in normal subjects by first occluding a limb with a cuff, then releasing the cuff; he thinks the hyperpnea due to stimulation of pressoreceptors in the pulmonary vascular bed by the sudden influx of blood from the periphery. Then, experiments performed by Harrison, Harrison and Marsh,[21] later confirmed by Megibow et al., according to Hoff, indicated that perhaps hyperpnea was due to a reflex arising from the great veins and right auricle; these workers induced hyperpnea in animals by inflating balloons in their superior venae cavae and noted the disappearance of hyperpnea upon vagal section. These observations are interesting since elevated venous pressure is a common, though not constant finding in congestive heart failure; however, hyperpnea and dyspnea are not exactly synonymous. Lastly, if heart disease results in a decrease in the oxygen tension of the plasma of the arterial blood (rarely), the chemoreceptors in the carotid and aortic bodies (located at the bifurcation of the common carotid artery and in the aortic arch, respectively) are stimulated and send afferent impulses via the vagus nerves which result in increase in rate and depth of breathing.[22]

The different clinical types of cardiac dyspnea commonly seen are dyspnea on moderate exertion, orthopnea, and cardiac asthma. Harrison et al.[23] showed that even in normal subjects proprioceptive impulses arising in the active muscles are transmitted to the respiratory center and result in increased breathing. It is well known that exercise increases the venous return, and it is also a fact that the increased depth of breathing, by increasing the negative intrapleural pressure within the thorax, favors venous return. Harrison states this results in an increase in venous pressure in the great veins; if the venous pressure in the cardiac patient is already elevated, then the dyspnea is of a more marked degree than would be seen in a normal individual following a similar amount of exercise.

Orthopnea is that form of dyspnea which is initiated or made worse by assuming the recumbent position. McMichael and Sharpey-Schaefer,[24] using right heart catheterization, proved that the cardiac output of each ventricle is increased upon lying down. In heart failure with the lungs already rigid from edema, the right ventricle must pump more blood into the lungs when the patient becomes recumbent, adding insult to injury by still further congesting the pulmonary tissues and leading to increased dyspnea via the Hering-Breuer reflex. There may be other contributing factors.

Cardiac asthma is that form of dyspnea which usually comes on at night in a patient whose lungs are already congested from heart failure, manifested by the patient's having to sit bolt upright in bed in order better to get his breath, his feeling of extreme suffocation and extreme anxiety, the presence of a chest full of bubbling rales, and, not infrequently, by the hawking up of frothy, blood-tinged sputum. The body position during sleep alone can lead to an

increase in pulmonary congestion; in addition, Harrison[13] thinks that other stimuli arising in various parts of the body may act as "trigger mechanisms" possibly via further increase in venous return and resultant increased right ventricular output in initiating the distressing syndrome; he lists these as: excess heat or cold, alcoholic intoxication, coughing, constipation, abdominal distention, desire to urinate, nightmares, and slipping down in bed.

Patients with left heart failure have decreased vital capacities. Christie and Meakins[25] found that the greater the dyspnea the less the vital capacity, and concluded that the reduction in vital capacity was due to the pulmonary congestion. Since, by definition, the vital capacity is that amount of air which, after the deepest possible inspiration, can be forced out of the lungs on maximal expiration, it is obvious that in dyspneic patients whose inspiratory excursions are cut short, the total amount of air capable of being exhaled is decreased.

Pulmonary congestion affects the intrapleural pressure. The intrapleural pressure during normal breathing at rest varies from about minus 2 to minus 6 mm. of mercury, the pressure becoming more negative upon inspiration—the greater the depth of inspiration the greater the increase in the negativity of the intrapleural pressure. In a patient with congestive heart failure and dyspnea, there is a marked increase in negative pressure which literally sucks more blood up from the periphery into the great veins and into the right heart, resulting in the right ventricle's gushing forth an additional volume of blood into the already congested lung and thereby further increasing dyspnea—a vicious circle is in progress, which if allowed to progress untreated, may result in the death of the patient by drowning in his own lung juice. But the marked increase in negative intrapleural pressure not only increases the venous return, but also may cause distention of the pulmonary capillaries and venous sinuses, which results in a pooling of the blood already present, thereby decreasing the flow through the pulmonary veins to the left ventricle. Cournand,[4] by means of right heart catheterization, has shown that in normal subjects deep inspiration results in an increased output by the right ventricle and a decreased output by the left ventricle; there is no reason to doubt that such similar effects occur in cardiac dyspnea, and when such obtain, the left heart receives less blood from the lungs than is ejected into them by the right heart, and pulmonary congestion is augmented.

That the velocity of the blood flowing through a congested and distended lung is slowed should be readily seen; a law of biophysics says, in effect, that the speed of flow varies inversely as the cross section of the vascular bed. This is borne out by the clinical observation that the arm to tongue circulation time is usually prolonged in cases of pulmonary congestion.

Pulmonary dynamics in congestive heart failure studied by means of right heart catheterization: The relatively recent perfection of the technic of slipping a small rubber catheter up the veins of the arm or leg and into the right side of the heart opens up new and fertile fields for the direct observation of not only the many physiologic phenomena inherent in the normal heart cycle, but also the alterations in intracardiac pressure and flow resulting from pathologic changes in the heart.

Although the use of this direct method of approach is still in its infancy, nevertheless, it has yielded some pertinent information regarding hemodynamics in congestive heart failure from various types of heart disease. Bloomfield et al.[2] observed in a patient in congestive heart failure having mitral, aortic, and tricuspid insufficiency on a rheumatic basis, that the mean auricular and right ventricular end-diastolic pressure was 29 mm. mercury and the right ventricular systolic pressure averaged 103 mm. mercury, or approximately four times the normal value; following compensation, the auricular and ventricular end-diastolic pressure returned to normal but the ventricular systolic pressure remained elevated. A patient in failure from arteriosclerotic heart disease had a right auricular

and ventricular diastolic pressure of 10.5 mm. mercury, and a maximum ventricular systolic pressure of 75 mm. mercury. Measurements in a man with decompensated syphilitic heart disease revealed right end-diastolic and high systolic ventricular pressures of 12 and 73 mm. mercury, respectively. In another case of rheumatic valvulitis, the authors recorded the pressures in both the pulmonary artery and the right ventricle: the maximum systolic pressure in the pulmonary artery was identical with that in the ventricle, measuring 83 mm. mercury; the diastolic in the lesser arterial circuit was 25 mm. mercury, or about three times normal. These investigators found that in all cases of left ventricular failure studied, regardless of etiology, the right ventricular systolic and pulse pressures (difference between ventricular systolic and diastolic pressure) were definitely increased. And they feel that an increase in right ventricular pressure points to a similar increase in pulmonary arterial pressure.

Richards[6] presented data on a patient with cor pulmonale, secondary to primary disease in the lung, in which the right ventricular systolic pressure rose to 60 mm. mercury. Figures on a case of mitral stenosis indicated a tremendous hypertension in the pulmonary circulation, as the maximum ventricular systolic pressure reached 120 mm. mercury, and only fell to 50 mm. mercury after clinical compensation was established. In this instance there was an apparent five-fold increase in the vascular bed, and one can see the tremendous importance of pulmonary congestion in left-sided heart failure.

Hickam and Cargill[3] studied pulmonary arterial pressures in patients in congestive heart failure both at rest and after exercise; some had mitral stenosis while others were victims of syphilitic and hypertensive heart disease. They found that the pulmonary arterial pressure was elevated in each patient before exercise and that it increased markedly after exercise; their exact figures are not available.

These studies by the several investigators indicate that patients exhibting clinical left-sided heart failure invariably have pulmonary hypertension, as reflected by abnormal elevation of the pressure in the pulmonary artery and right ventricle.

Pathogenesis of pulmonary congestion: If we offer a correct solution to the cause of pulmonary congestion, we will, of course, solve the problem of the pathogenesis of clinical left-sided heart failure, for the dysphea, orthopnea, and fine moist rales in the lungs present in so-called left-sided failure are due to the pulmonary congestion, as has been shown above. But has there ever been offered a perfectly sound and scientifically proved explanation of pulmonary congestion—that it is due to one solitary factor, or that there are several factors responsible, each of which is consistently altered in a similar manner in every case of left-sided heart failure? Not that I am aware of. But let's follow a case of, say, essential hypertension and see how pulmonary congestion may result. The work that the left ventricle does may be expressed as a product of its output times the peripheral resistance; when the latter is increased by essential hypertension, the left ventricle must work harder to maintain a normal output. As a result of this extra work, the ventricle undergoes hypertrophy and dilatation, but it is still efficient, if its efficiency can be measured by its ability to continue its usual output. A critical point is reached finally, however, when the ventricle cracks and it is physically unable to dispatch all the blood which it receives. It is more or less generally accepted that this inability of the ventricle to fully discharge its contents is the first step toward full-blown left-sided failure.[26] As residual blood accumulates and the left ventricle further dilates, its pressure during diastole rises,[16] making the left auricle's job of passing its blood into the ventricle more difficult; but by hypertrophy and dilatation it performs its task efficiently for a while then, like its partner, it tires and is unable to pass forward all of the blood coming from the lungs. When, as a result of the residual blood in the left auricle, the intra-auricular pressure

rises, and acts as a barrier to the blood coming from the lungs via the pulmonary veins, there must be a damming back of blood into the lungs. To store this blood which the right ventricle puts out but which the left side of the heart is unable to relay, additional capillaries open up throughout the lungs which later dilate as more blood accumulates. These events keep the pulmonary intracapillary pressure from rising too high, but when there are no more capillaries available to absorb the excess blood when the ones in use are dilated to their maximum, then of necessity, there must be an additional increase in intracapillary pressure.

Now, granted that there is increased intracapillary pressure secondary to left auricular failure, is that pressure from this source alone enough to drive fluid across the capillary membrane into the parenchyma of the lungs? Landis and Gibbon[27] showed that fluid will pass out of the capillaries into the tissue spaces if the venous pressure rises above 100 mm. water. But there may be another factor which causes a still further elevation of the intracapillary pressure. It is a fact that most patients in congestive heart failure have an increased blood volume[16]; it is not illogical to reason that the presence of still more fluid in the engorged capillaries further increases the intracapillary pressure up to a point where there occurs transudation of fluid into the lungs, causing pulmonary congestion and the clinical picture of left-sided heart failure.

There have been several explanations as to why the blood volume is increased. Futcher and Schroeder[28] observed that the renal excretion of sodium chloride was markedly reduced in four patients in congestive heart failure. Warren and Stead[29] conclude that in congestive heart failure there is a disturbance of renal function, as a result of which salt and water are retained in the body and produce an increase in blood volume. Merril[30] states that Lyons showed that salt and water retention can produce a rise in venous pressure in normal subjects. Grant and Reischman[31] demonstrated an increase in blood volume, venous pressure, and volume of extracellular fluid in healthy medical students following ingestion of an excess of sodium chloride. So it seems that the retention of salt (the sodium ion in particular) in the body can be a major factor in the production of an increase in blood volume in both normal subjects and in those in congestive heart failure. The details of the theory of the mechanism of sodium retention will not be gone into here; the reader is referred to papers by Futcher and Schroeder,[28] Warren and Stead[29] and Merril.[30]

That derangement of salt and water metabolism is a factor of major importance in considering the pathogenesis of pulmonary congestion in heart disease is borne out by clinical observations. Many investigators [20, 29, 30, 32, 33] have found that patients with severe congestive heart failure can be made to lose their gross peripheral and pulmonary edema and become clinically compensated simply by rigid restriction of salt intake and without the use of digitalis; further, once apparently compensated these same patients can be thrown back into heart failure at will by the excessive ingestion of salt and water, even if digitalis is given concurrently; the return of pulmonary and peripheral edema, dyspnea and orthopnea is usually preceded by a rise in blood volume.

In spite of the fact that increased blood volume often accompanies, and commonly even precipitates, the development of cardiac pulmonary congestion, one can by no means say, therefore, that it is the sole or most important cause of this phenomenon. Before marked engorgement and actual pulmonary edema fluid forms, there first must be an increase in pressure transmitted backward from the left auricle to the pulmonary veins and into the lung capillaries; after the stage is set, so to speak, then the additive effect of any increased blood volume in further raising the intracapillary pressure may be sufficient to initiate or increase a transudation of fluid across the capillary walls into the parenchyma of the lung.

Observations made in the course of ex-

perimental work on animals certainly suggests that many types of pulmonary edema, even cardiac, may be partially reflex in origin. Henneman[14] presents an excellent review of the subject of the experimental production of acute pulmonary edema, describing in detail some of the interesting procedures; the data below are taken from Henneman's paper. Antoniazzi produced acute pulmonary edema in rabbits by ligation of the arch of the aorta disal to the origin of the carotid arteries; the edema could not be produced if both vagi were sectioned previously; the central nervous system was easily depressed by hypnotic drugs to a point where no edema resulted after aortic ligation. Luisada, after experimenting with 250 rabbits and producing pulmonary edema by intravenous adrenalin, felt that the edema resulted from a hypertension produced in the carotid and cephalic vessels. The conception that pulmonary edema following phosgene poisoning is due to direct action on the lung capillaries was challenged by the observation that if the vagus nerves were cut previously the classical signs and symptoms were largely prevented. In more recent experiments than those cited by Henneman, Luisada and Sarnoff[31] noted that they could produce acute pulmonary edema in dogs by the rapid infusion of fluid into their common carotid arteries, but could not produce edema when the same amount was injected at a similar rate into the femoral arteries; they found also that if the carotid sinus were denervated, pulmonary edema failed to occur in five out of seven dogs. They believed that ". . . . . the results of experiments on pulmonary edema caused by adrenalin, ligation of the aortic arch and venous infusion. should now definitely focus the attention of students of pulmonary edema upon the nervous system and not upon the myocardium. That either irritation of the myocardium (coronary occlusion), or distention of the heart cavities (heart failure, hypertension) may be one of the causes of pulmonary edema cannot be denied, but we feel that either of these phenomena is secondary to the distention of the large arterial vessels as a cause of reflexes which favor acute edema of the lungs. For the occurrence of pulmonary edema a condition of distention of the lung capillaries is essential. On this condition, an acute reflex increase of permeability may be superimposed which will lead to paroxysmal pulmonary edema."

SUMMARY AND CONCLUSIONS

1. The pulmonary dynamics in some of the more common congenital cardiac anomalies are discussed, including data derived from right heart catheterizations in these conditions.

2. Patients with valvular heart disease have abnormalities of cardiac function which often may be detected prior to the onset of left-sided heart failure.

3. Pulmonary congestion is discussed from the standpoint of pathologic anatomy, pathologic physiology and pathogenesis.

4. Observations on intracardiac and pulmonary arterial pressure changes in left-sided congestive heart failure as recorded by direct right heart catheterizations are reviewed.

5. Patients in right-sided congestive heart failure have hypertension in the pulmonary vascular bed manifested by increased pressure in the pulmonary artery and right ventricle.

6. Pulmonary congestion of cardiac origin depends upon increased pulmonary intracapillary pressure, this resulting primarily from an elevated venous pressure subsequent to failure of function of the left auricle but augmented by an increased blood volume. There is evidence to show that reflexes arising directly from the heart, the great vessels and some of their main branches, may play a very important contributing role in the production of pulmonary edema of cardiac origin.

7. There are probably other factors as yet poorly understood concerned in the production of pulmonary congestion.

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THE WOLFF - PARKINSON - WHITE SYNDROME (BUNDLE OF KENT SYNDROME; FALSE BUNDLE BRANCH BLOCK)*

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NEW ORLEANS

When Wolff, Parkinson and White[35] presented the first comprehensive report of this syndrome in 1930, they evoked a world-wide response. The interest in this electrocardiographic pattern has even now not subsided, principally because of the controversy concerning its mechanism and prognosis. The literature abounds with case reports, interpretations and conflicting data and opinions. In this paper I shall attempt to present in a succinct manner the most authoritative and widely-held views and to report a series of cases gleaned from the files of the Charity Hospital in New Orleans.

DEFINITION

Although cases were reported by Wilson[32] in 1915 and again by Wedd[31] in 1921 and by Hamburger[9] in 1929, it was not until 1930 that a clinical entity was evolved, for a that time Wolff, Parkinson and White[35] collected and reported eleven cases with careful four year follow-ups.

The electrocardiographic tracing is composed of a short PR interval with a widening of the QRS complex in such a manner that the time from the beginning of the P

*Awarded the prize given by the Professor of Medicine at Tulane University School of Medicine to a Senior student who presented the best thesis on a subject related to internal medicine.

wave to the end of the QRS complex is within normal limits. The PR interval is usually less than 0.1 second and the QRS complex has characteristics suggesting bundle branch block. The ventricular complexes are wide and slurred, and the T waves may be opposite in direction from the main QRS deflection. The most usual type simulates a left bundle branch block, with the initial ventricular complexes as seen in left axis deviation and the T waves in leads I and III in the opposite direction from the main deflection of the QRS complex. If it were not for the short PR intervals, these tracings would be indistinguishable from organic bundle branch block.

INCIDENCE

To date, approximately 300 cases have been reported in the literature; obviously the actual number of cases is far greater. Ohnell,[19] in his comprehensive monograph on this subject, reports an incidence of from 0.05 to 0.1 per cent. Boyer,[2] in a review of 2000 tracings of 1356 patients in the Army, reports an occurrence of 0.5 per cent in this group. This phenomenon is usually manifested in the young (second, third and fourth decades), most commonly in the male, perhaps accounting for Boyer's high figures among soldiers.

CLINICAL DATA

Frequently the history dates back to puberty, when the patient first began to experience periodic attacks of palpitation after exercise or even at rest, but this finding is not constant. Of the arrhythmias which these patients are prone to develop, paroxysmal, supraventricular tachycardia ranks first; auricular fibrillation occurs with considerably less frequency. Lately, a few cases have been reported[18] in which there were paroxysms of ventricular tachycardia.

In many cases, both the PR interval and the QRS complexes suddenly revert to normal without change in direction or contour of the P waves. This may occur spontaneously or when the heart rate is increased by exercise or atropine. When the heart rate slows, the abnormal characteristics may return. Patients in this group are particularly liable to attacks of paroxysmal tachycardia during which the ventricular complexes invariably* assume a normal configuration.

It has been shown[11] that there is a greater incidence of paroxysmal tachycardia in those patients with QRS changes as well as short PR intervals. It is of interest to note that among patients exhibiting the phenomenon of paroxysmal, supraventricular tachycardia, 5 per cent demonstrate a Wolff-Parkinson-White type electrocardiogram, and that conversely among patients recording this tracing 70 per cent are subject to paroxysms of supraventricular tachycardia.[19]

MECHANISM

Wolff, Parkinson and White originally suggested that the mechanism was based on increased vagal tone, implying that there is a paradoxical vagal effect with a simultaneous exercise of two diametrically opposed influences, one accelerating conduction between auricles and ventricles with shortening of the PR interval, the other retarding conduction through the bundle of His, giving rise to the lengthening of the QRS complex. This concept is mentioned principally from the historical point of view since it is now usually considered untenable, in the light of recent evidence, by most observers. White,himself, is now supporting the theory of accessory bundle (*v. infra*); on the other hand, Parkinson together with Hunter and Papp[11] hypothesize that the anomalous beat arises near but not within the sinoauricular node, and that the normal ventricular complex is interfered with by a ventricular extrasystole which arises prematurely and low in one bundle branch.

Today, the most widely held, although by no means universally accepted theory is the hypothesis of an accessory pathway of atrioventricular conduction which was first introduced by Holzman and Scherf[10] in 1932 as a possible explanation. Almost simultaneously Wolferth and Wood[33] advanced the same hypothesis which they substantiated

*Movitt (18) reported a case in which the aberrant bundle (*vide infra*) was evidently able to conduct the auricular impulses during supraventricular tachycardia, leading to preservation of the aberrant QRS complex during the attack.

histologically in 1943.[36] Since this theory is the one for which there is the best supportive experimental data, and the one which lends itself to the clearest thinking in dealing with this phenomenon, we will confine our discussion to it. For a comprehensive survey of all the theories ever promulgated as an explanation of this subject, the reader may refer to Ohnell's monograph on preexcitation.

More than 30 years ago Kent demonstrated histologically a muscle bundle in the atrioventricular groove which joined the right auricle and right ventricle in man. He termed this structure the "right lateral bundle." Interest in his finding lay dormant until the advent of the electrocardiographic anomaly under discussion, when in 1933 Wolferth and Wood utilized it in explaining the mechanism of this pseudo-bundle branch block.

When in 1933, Wolferth and Wood[33] presented the theory of a functioning, conducting right lateral bundle, they reported nine cases of this syndrome which could not be reverted to normal with exercise or atropine. In presenting their hypothesis they reasoned as follows:

1. The short PR interval is due to the short, direct pathway from auricle to ventricle via Kent's bundle.

2. The premature invasion of the ventricular myocardium by this impulse causes: (a) a slurring of the initial QRS deflection and (b) a widening of the QRS complex at the expense of the PR interval.

3. In some cases, conductivity in the bundle of Kent may not be highly developed. Consequently, when subjected to the passage of impulses at a rapid rate, as during paroxysmal tachycardia, it may fail to function.

4. Kent showed that retrograde conduction in rats was possible under certain circumstances. Therefore, a retrograde impulse might travel from ventricle to auricle at a time when the physiological state of the auricular muscle would favor the inception of an abnormal rhythm. This mechanism might explain the frequency of paroxysmal tachycardia in these people.

5. The assumption of a functioning bundle of Kent does away with the necessity of postulating a lesion or defect in conduction in youthful patients without evident cardiac damage.

6. During both normal and abnormal mechanisms, the duration of the interval from the beginning of the P wave to the end of the QRS complex tends to remain constant. Only one interpretation seems possible; namely, that the variable factor is early aberrant conduction from auricle to ventricle. This does not interfere with conduction through the junctional tissues, but alters the duration and form of the initial portion of the ventricular complex in direct proportion to the prematurity of arrival of the aberrant impulse in the ventricular myocardium.

Then in 1943, these same authors[36] studied a case of typical Wolff-Parkinson-White syndrome in a young boy subject to attacks of paroxysmal tachycardia, but in whom no evidence of heart disease was ascertained. The typical tracing was a constant finding. He died at the age of 16, two hours after an attack of paroxysmal tachycardia.

At autopsy the heart was grossly normal and there was no accountable lesion. They made serial sections about the atrioventricular groove beginning just to the right of the aorta. In this manner they demonstrated a muscle bundle leaving the auricular muscle and joining the ventricular muscle after passing by means of a bridge of tissue across the ventricular cavity just below the attachment of the tricuspid valve. They demonstated in this patient's heart three such accessory connecting bridges of muscle tissues between the right auricle and right ventricle, these connections being of the type which should be able to conduct an impulse from auricle to ventricle. This study supports the hypothesis of accessory pathway conduction. Proof would require direct evidence that these tracts are capable of transmitting the excitory process. The question of the incidence of these accessory bundles is also unanswered.

Further substantiating evidence for this

theory is recorded by Rosenbaum, Hecht, Wilson and Johnston[24] wherein they show that the form of the ventricular complex in unipolar leads from the esophagus, precordium and other points on the thorax suggests that in these cases the dorsal wall of the ventricles is activated prematurely by impulses of supraventricular origin. They also find evidence that the atrioventricular node and bundle continue to function normally in this condition. They also note that the anomalous ventricular complex assumes the normal form when the pacemaker shifts to the lower levels of junctional tissue. These observations support the view that impulses pass from the auricles to the ventricles not only by the normal route but also by one or more additional channels.

Finally, the work of Butterworth and Poindexter[3] should not go unmentioned. They demonstrated that a typical Wolff-Parkinson-White pattern could be reproduced in animals by the artifical introduction of a short circuit comparable to the right lateral bundle, lending further support to the hypothesis of premature aberrant conduction.

In short, then, there are good experimental data and clinical observations to point out that accessory pathway conduction is the probable mechanism at work here. At any rate it offers the most satisfactory working hypothesis.

PROGNOSIS

What do we tell the patient in whom a Wolff-Parkinson-White syndrome is demonstrated? Is it necessary to say anything, or are we dealing with a phenomenon of no clinical signficance? Indeed, we are faced with the dilemma of perhaps creating a cardiac neurosis, once established difficult to dislodge, or on the other hand of performing an injustice to the patient by not informing him of a hazard to his chance for a long life. My only contribution to this problem must be that of faithfully recording both sides of the controversy.

Lynch and McAllister[16] present the view that we are dealing with a functional phenomenon and that the patient is suffering from an electrocardiogram rather than a disease. They would ignore the finding and give the patient complete assurance for life and unrestricted activity on the basis of the cardiac status of the moment.

Bishop[1] along the same line of thought brings up the argument that when heart disease is an associated finding with the syndrome it is of such diversified forms that it is hardly conceivable that the organic heart disease when encountered is more than coincidental. He reiterates the common observations that exercise does not seem to increase the frequency of the attacks, for they may well occur at rest and that apparently, a small percentage of patients seem to outgrow their "electrocardiographic difficulty" without first decreasing their activity.

Boyer,[2] relating his Army experience with this problem, finds only an abnormally increased incidence of psychoneurosis in these patients, emphasizing, of course, the importance of caution when offering these patients a guarded prognosis.

On the other hand are the many reports of the association of this pattern with such entities as active rheumatic heart disease, aortic regurgitation, mitral stenosis, hypertensive heart disease and thyrotoxicosis.[11, 28] Master, Joffes and Deck[17] report a case with thyrotoxicosis without heart disease in which the pattern reverted to normal after thyroidectomy.

In referring to this syndrome in an address[37] to a group of insurance medical directors, Frank Wilson remarked that he did not consider this condition trivial. He recounted one patient who had died with prolonged tachycardia, and at autopsy definite evidence of myocardial change was found.

Fox[7] suggests that it is conceivable in the older age group that a circulatory difficulty of the A-V node due to coronary sclerosis may sufficiently depress its functional activity to permit the aberrant mechanism to take over some of the conducting function.

Kimball and Burch[13] have collected six deaths from the literature and reported two more cases among which at least five were attributed to paroxysmal tachycardia associated with the Wolff-Parkinson-White

syndrome. Three other deaths were said probably to be due to paroxysms of tachycardia in patients with a typical picture of the syndrome. Their opinion is that the Wolff-Parkinson-White syndrome should be regarded as strongly suggestive of definite evidence of heart disease. They suggest that a guarded prognosis be discussed with a responsible member of the family rather than with the patient in order to avoid precipitating a cardiac neurosis.

THE WOLFF-PARKINSON-WHITE SYNDROME IN THE CHARITY HOSPITAL IN NEW ORLEANS

In an attempt to determine how a local series of patients manifesting the Wolff-Parkinson-White electrocardiogram would correlate with the data accumulated in the literature, I selected patients who had been reasonably well worked up and from whom at least one typical tracing had been recorded. There were ten known patients of this description in the files of the Charity Hospital record library.

Incidence: All but one of these patients were between the ages of 19 and 55 years and 60 per cent (six patients) were less than 37. There were eight white patients, two colored patients, six females and four males. One colored patient was a male and the other a female.

Heart Disease: Two patients had no determinable evidence of heart disease. Another patient probably had some cardiac changes as the only typical tracing was obtained when she was in profound uremia which terminated fatally two months later. It is not known whether or not a fourth patient had heart disease, as the typical tracing was recorded as a routine procedure before metrazol shock therapy was instituted for hebephrenic schizophrenia.

The other six patients had definite evidence of organic heart disease. It is well to bear in mind at this point that these tracings were not recorded routinely, but rather ordered in patients in whom a determination of cardiac status was indicated for one reason or another. For example, in this group of patients there were five hypertensives, three patients with definite signs of failure, and four patients with enlarged hearts. Therefore, the presence or absence of heart disease in these patients is not very significant. However, the types of heart disease encountered is important. That is to say, we would like to know if this electrocardiographic tracing is common to any particular type or group of heart diseases. Accordingly, a resumé of these cases and the type of heart disease occurring in them becomes essential.

1. A young, white male suffered a metapneumonic empyema in 1935, complicated by purulent pericarditis which was drained, leaving him with an adhesive pericarditis. Five years later two typical Wolff-Parkinson-White tracings were recorded. These were found to be of a normal mechanism, but recorded at a time of a much higher pulse rate. In the five year interval he suffered six attacks of paroxysmal tachycardia and occasional bouts of dyspnea on exertion, but no other difficulties.

2. A young colored male was in the psychiatric ward with a diagnosis of hebephrenic schizophrenia. A routine electrocardiogram revealed the anomalous tracing. It was regarded as normal and no further cardiac studies were made. It is not known whether he suffered attacks of tachycardia.

3. An elderly white female had hypertension, dyspnea, orthopnea, ankle edema, tachycardia, precordial pain and an enlarged heart. She actually developed a paroxysm of tachycardia while on the ward. Her tracing was somewhat equivocal in that the PR interval was somewhat longer than that usually seen. There was no repeat tracing.

4. A 37 year old white female was a diabetic with hypertensive cardiac disease. She had an enlarged heart, weakness, dyspnea, ankle edema and precordial pain. Her tracing was not completely typical in that the QRS complex was slurred but not of long duration. The PR interval was .1 second, and the heart rate was 121. An earlier tracing had a normal mechanism at a heart rate of 102.

5. A 53 year old white female had a typical tracing recorded while she was in the hospital for management of her diabetes. There was no history of tachycardia or

symptoms of heart disease, nor was there any evidence of heart disease by physical examination.

6. A 55 year old white male had severe hypertensive, arteriosclerotic heart disease. He had many admissions into the hospital for congestive failure. He was in auricular fibrillation for two months, finally returned to a normal mechanism and was discharged. A year and a half later he was admitted again in congestive failure when an electrocardiogram was taken. The tracing was thought to show Wolff-Parkinson-White patterns in that the PR interval was .13 second and the QRS .14 second with a slurred and notched R_1. This was the only tracing demonstrating this mechanism.

7. A young white female was admitted for a normal pregnancy. An electrocardiogram was taken because of a history of paroxysmal tachycardia. The tracing was a typical pattern of pseudo bundle branch block. She was normotensive, but had occasional bouts of weakness and orthopnea. She had cardiac enlargement. The tracing was repeated the next day and showed the same mechanism.

8. This patient was another young white female who was in uremia on the basis of malignant nephrosclerosis. One tracing demonstrated a typical pattern of aberrant conduction. The next tracing taken three weeks later showed a normal mechanism. Five weeks later she expired.

9. A 34 year old white male was admitted for multiple injuries sustained in an automobile accident. An electrocardiogram made on that admission was interpreted as supraventricular tachycardia with a heart rate of 240 beats per minute. He was discharged the same day, to return a few days later with evidence of increased intracranial pressure. An electrocardiogram taken on this admission demonstrated a Wolff-Parkinson-White pattern. He had a past history of occasional attacks of tachycardia before his accident. There was no evidence of heart disease.

10. A 55 year old white female had severe hypertensive cardiovascular disease for 12 years. One electrocardiogram showed a typical Wolff-Parkinson-White syndrome with a 2:1 block. A later tracing showed a return to a normal mechanism. There was no history of tachycardia.

In summary it is noted that in this group of patients a Wolff-Parkinson-White syndrome was associated with four cases of hypertensive heart disease, one arteriosclerotic heart disease, one severe uremia and one patient with adhesive pericarditis.

Paroxysmal Tachycardia: There was a history of paroxysmal tachycardia in four of these patients. It is not known whether or not a fifth patient (the schizophrenic) had a history of tachycardia.

Of this group of patients then, we note that there is a high incidence of associated heart disease, especially of hypertensive etiology; this is explained in that this was not a routine series of electrocardiograms, and that hypertension is a common factor in heart disease in the population of which this group of patients is representative. As might be expected, there is a high incidence of paroxysmal tachycardia in the group. The distribution of these patients by age and sex is comparable to that reported by other investigators.

SUMMARY

1. The Wolff-Parkinson-White syndrome is defined as the combination of an electrocardiographic pattern of a short PR interval, wide, slurred QRS complex and a normal P-J interval in a young patient who commonly demonstrates paroxysmal tachycardia.

2. The mechanism of this pattern is probably that of accessory pathway atrioventricular conduction with short-circuiting of the excitory wave. This theory is explained and the evidence in favor of it is presented.

3. The actual significance of this finding is as yet undetermined. Many are inclined to dismiss the syndrome as being without clinical importance, but some reported deaths are herein discussed.

4. A series of cases from the Charity Hospital is reported, and discussed.

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PUBLIC RELATIONS PROGRAM OF LOUISIANA STATE MEDICAL SOCIETY

The Council on Medical Service and Public Relations of the Louisiana State Medical Society recently announced its Public Relations Program for the year 1948. The program comprises eleven major parts and is to be promulgated under the leadership of the Council in cooperation with the Executive Committee and the House of Delegates of the Louisiana State Medical Society and will serve as a guidepost to the component parish medical societies throughout Louisiana. Dr. A. V. Friedrichs, Chairman of the Council, made the announcement.

By the institution and fulfillment of the program the doctors of Louisiana intend to overcome the propaganda issued by the Government relative to a compulsory system of medical care. Through a program of progressive accomplishments by the medical profession, rather than through a program of expensive propaganda as indulged in by the Government, it is agreed that the doctors have the answer to the proponents of socialized medicine.

One phase of the program, namely, "General Public Relations", provides for the presentation of educational health radio programs, especially in the rural areas of this state. A speakers' bureau is to be established and lay persons and members of the profession will appear before civic clubs, women's clubs, parent-teacher groups, and other interested groups to present educational health talks, talks on economics of medicine, voluntary health insurance, and numerous other subjects.

An educational health column is to be released weekly and it is anticipated that it will appear in all newspapers throughout Louisiana. A news bureau will be established to disseminate pertinent information to the general public relative to the scientific advancements made by the profession and also to advise the general public what the medical profession is doing in their behalf.

Another part of the program is to promote voluntary health insurance. The Louisiana Physicians Service is operating the "Blue Shield" plan, which offers surgical and obstetrical care and is sponsored by the Louisiana State Medical Society. "Blue Cross" hospitalization coverage is also part of the voluntary health program. Through this medium persons who are willing to voluntarily enroll may contribute a monthly fee and thereby prepay the costs of the doctor bill for surgery and hospitalization.

It is proposed that the Medical Society will take leadership and assist in the estab-

lishment of health councils in every parish throughout the state in cooperation with all other voluntary agencies interested in health. Another phase of the program includes a closer working relationship with other health agencies, societies, and civic clubs, and a program and medium to keep cooperating government health agencies adequately informed of voluntary health progress.

The program also includes the institution of a campaign to maintain and enhance patient-physician relations.

In general, the program provides for a medium to maintain adequate medical care at a cost which the patients can afford and at the same time keep medicine a private enterprise.

In times past the position of the physicians and the appreciation of the public left no question as to how medicine should be practiced. Now the doctors have to fight to maintain this position. Active participation by the membership is needed.

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LOUISIANA PHYSICIANS SERVICE, INC.

A vital contribution of the physicians of Louisiana in the fight against state medicine is the Louisiana Physicians Service, Inc. The Louisiana Medical Society, as does organized medicine in many states, sponsors a voluntary sickness insurance plan which has been in operation since January 1, 1947. This is a necessary part of our plan to meet the criticism that medical care is so expensive that it is inaccessible to the majority. In all of the schemes of the social planners it must still be remembered that the public ultimately pays for medical care regardless of the method by which payment is collected. We wish to continue to show that medicine as a private enterprise, with voluntary insurance, is best.

Securing proper adherence of the public to any plan is largely dependent upon the support which the individual physicians give it. When the physician indicates that he is participating, he renders support to the profession. Insurance of this type has little statistical experience to guide it. Our organization is courageously furthering the interests of voluntary insurance and necessarily of the individual member. The aim therefore is that all should familiarize themselves with the details, should participate in the surgical agreement and should, above all else, acquaint their patients with the advantages.

There are now 20,351 individuals enrolled on our plan. There are 64 plans in operation over the nation. The Louisiana Physicians Service, Inc. is the 46th in size over the country, having surpassed some 18 plans in growth in the first year. The board and administrators of Louisiana Physicians Service, Inc. are to be congratulated on their achievements thus far. Our own protection depends on our strenuous collective effort.

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THE BLOOD BANK SITUATION

It is learned that recently the American Red Cross has undertaken a National Blood Bank Program designed eventually to provide blood and blood derivatives to the entire nation without charge for the products. As is usual in matters where nationalization of medicine is in view, the figures are astronomical. The projected plan is to spend $5,000,000.00 to start the program and $20,000,000.00 annually; it is reported that 3,700,000 pints of blood must be obtained within the next year—one pint for every 35 Americans.

The American Red Cross, while not politically dominated, operates with government sponsorship; not with tax money but with public subscription. The allotment of blood and its products by the American Red Cross would ultimately lead to the effect of having the Red Cross practice medicine. The transition from this arrangement to state medicine could become an imminent danger.

The physicians who operate the blood

banks of hospitals and communities have formed an association. The association is composed of institutional and individual memberships. Their objective is to pool their knowledge and influence; to render useful and adequate service. It seems fitting in the interest of good medicine and the ultimate welfare of the patient, that operation of blood banks be kept in the hands of those who have it now and of this association.

Medicine did not grow up to the magnificent cooperative effort it is now by the work of social planners and bureaucratic directives. It is the work of physicians, striving together for the common good. The needs are not too big for such cooperation. Jefferson said "Government governs best which governs least."

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

PRESIDENT ANDERSON

The President of the Louisiana State Medical Society, Dr. Gilbert C. Anderson, completes his term of office in April, 1948. As a marker along the long road of service he has travelled for organized medicine, we take pleasure in presenting a brief biographical sketch.

Dr. Anderson was born in Jackson, Tennessee, educated in the public schools and graduated by Southwestern Baptist University, of that city, with the degree of Bachelor of Arts. Doctor of Medicine was granted by the College of Physicians and Surgeons of Columbia University and he served as intern in the City Hospital and House Surgeon in the New York Hospital, both of New York City.

During the first World War he entered the United States Army as first lieutenant and was immediately assigned to neurological surgery at which he worked at Camp Greenleaf and General Hospitals Nos. 11 and 41.

Soon after discharge two very important and far reaching factors influenced his life; the first his marriage to Mildred Hodges Moore of Norfolk, Va. and the second an intensive postgraduate course in neurological surgery as a Fellow at the Mayo Foundation during the last year of which he was first assistant to Drs. Adson and Craig. For work done during and in connection with this Fellowship he was awarded the degree of Master of Science (in Surgery) by the University of Minnesota.

Upon completing his fellowship he came to New Orleans and entered the practice of

his specialty late in 1927. He was appointed on the Faculty of Tulane University which post he held until the founding of the Medical Center of Louisiana State University where he accepted the appointment which he now holds. Very soon after his arrival in New Orleans he aligned himself with organized medicine and has been active in that field of endeavor until the present time, filling various posts from time to time including the Presidency of Orleans Parish Medical Society. He is on the staff of several hospitals in New Orleans, is a member of local, sectional and national societies and a diplomate of the American Board of Neurological Surgery.

Following discharge from the U. S. Army after the first World War he accepted a commission in the Federalized Guard of the State of New York and was assigned to the 71st. Infantry Regiment; upon moving to Minnesota he joined the Organized Reserve and was very active in that work until the outbreak of the second World War which found him a Lieutenant Colonel and acting Colonel in command of the 312th. Medical Regiment, and Division Surgeon of the 84th Division, Organized Reserve; he reported for active duty and was discharged for physical reasons but worked during the war for Selective Service as a Member of Medical Reference Board. He also served as Lieutenant and Captain in the Civil Air Patrol and as Lieutenant Colonel and Chief Medical Officer of the Louisiana State Guard.

His hobby is his camp at Grand Isle.

The past year the administration of the affairs of the State Society has been a difficult task. Problems involving vital interests of the Society have been numerous. The ever-increasing battle against political medicine has had to be fought. The program of promoting solidarity in our ranks and of improving our public relations has been effectively supported. The Society is fortunate in having enjoyed the good effects of its president in so critical a year.

When Dr. Anderson relinquishes the presidency of the Society, it is not felt that he is retiring from an active part in the affairs of organized medicine but that he is merely joining the distinguished group of past presidents whom the Society has been proud to honor and upon whom it depends so heavily for advice, help and material service.

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ANNUAL MEETING

We wish to call your attention to the approaching meeting of our State Society in Monroe, April 12, 13, and 14th; headquarters in the Frances Hotel. Those who have not yet secured reservations should at once contact Dr. J. W. Cummins, who is chairman of the Hotel and Meeting Rooms Committee. The Annual Meeting this year will be most representative, and we are sure, one of the largest ever held. It will include an impressive scientific program and this should be an excellent time to review the scientific progress of medicine and surgery. It will also afford an opportunity to meet many distinguished colleagues. Many of the speakers are men of national and international reputation who have demonstrated marked proficiency in the respective subjects assigned to them. The following is the synopsis of the meeting:

Monday, April 12

Morning and afternoon: Meeting of House of Delegates; noon: Luncheon for House of Delegates; evening: General Public Meeting. This will probably be held at the Neville High School Auditorium and the speakers of the evening will be Dr. Morris Fishbein, of Chicago, and Dr. James E. Ayre, of Montreal. The meeting will be an open forum on the subject of Cancer.

Tuesday, April 13

Morning and afternoon: Scientific Program; noon: Luncheon for Members; evening: President's Reception.

Wednesday, April 14

Morning and afternoon: Scientific Program; noon: Specialty Luncheons.

Your attention is particularly called to the out-of-state speakers. Dr. Ira T. Nathanson of Boston, Mass.; Dr. Harry S. N. Greene, New Haven, Conn.; Dr. J. E. Faber, Rochester, Minn.; Dr. F. R. Lock, Winston-Salem, N. C.; Dr. Wm. F. Guer-

riero, Dallas, Texas; Dr. W. Ambrose McGee, Richmond, Va.; Dr. Herbert Mantz, Kansas City, Mo.; Dr. Jos. M. Hayman, Jr., Cleveland, Ohio; Dr. George B. Eusterman, Rochester, Minn., and Dr. James E. Ayre, Montreal.

It is expected that serious thought and attention will be given to this splendid scientific program that has been arranged. The doctors of the Ouachita Parish Medical Society and their friends in Monroe are doing everything in their power to make your visit in this city a pleasure, and a profitable program and suitable entertainment is being provided for everyone.

Following are copies of communications received from the Mayor of Monroe and the Secretary of the Chamber of Commerce, which are evidence of your welcome to the City of Monroe.

February 14, 1948

Dr. P. T. Talbot
New Orleans, Louisiana
Dear Dr. Talbot:

It is my understanding that the Louisiana State Medical Society Annual Convention is to be held in Monroe, Louisiana, April 12-14, 1948.

Monroe is a beautiful city and our people are known for their hospitality. We are indeed proud that your organization chose to have the convention here and we will do all we can to make it a success.

Please feel free to call on us if there is any assistance we can render.

Yours very truly,
H. H. BENOIT, *Mayor.*

February 13, 1948

Dr. W. L. Bendel
Wright-Bendel Clinic
Monroe, Louisiana
Dear Dr. Bendel:

We are delighted to learn that the Louisiana State Medical Society is to hold its Annual Meeting in Monroe, beginning April 12. You may rest assured that your colleagues in the medical profession and their wives will receive a very warm welcome from the citizens of Monroe.

If this organization can assist you in any way in making arrangements for your convention, we wish you to feel free to call on us. We extend to the Medical Society our best wishes for a most successful convention.

Sincerely yours,
H. G. BAILEY, JR., *Manager,*
Chamber of Commerce.

The tentative program of the Woman's Auxiliary which will provide wonderful entertainment for all the visiting ladies will be found in the Woman's Auxiliary section of this issue.

The following is a list of the committees and chairmen:

General Chairman: John G. Snelling.

Advisory Committee: Dr. John G. Snelling; Dr. A. Scott Hamilton; Dr. C. P. Gray, Sr.; Dr. J. Q. Graves; Dr. George W. Wright, and Dr. DeWitt T. Milam.

Badges: Dr. F. P. Rizzo advises that appropriate badges will be furnished at the Registration Desk.

Banquet: Dr. A. V. Miller, Jr. is in charge of all banquet arrangements.

Commercial Exhibits: Dr. Clifford Johnson reports that he is receiving the contracts from commercial exhibitors and will allocate the proper space and will do all in his power to assist in the exhibits.

Entertainments: Dr. C. B. Flinn is active in completing all necessary entertainment for our visitors.

Budget and Finance: Dr. Henson Coon will make disbursements as required and is receiving checks daily.

Golf: Dr. Fred Marx will stage a tournament, the time to be announced later and suitable prizes awarded. Bring your clubs and other paraphernalia.

Hotel and Meeting Rooms: Dr. J. W. Cummins suggests that you make reservations as early as possible and will select halls and rooms at headquarters for the various programs.

Lanterns and Projectors, Sound Equipment: Dr. M. W. Hunter has both a slide machine and motion picture machine in readiness, and will arrange for an operator.

Program: Dr. Glen Gallaspy has charge of the program and will endeavor to see that it is carried on at proper time.

Registration: Dr. P. L. Perot will see that the registration desk is in a conspicuous place on the Cherokee Terrace.

Luncheons: Dr. B. J. Lecour promises to have enough for everybody to eat.

Scientific Exhibits: Dr. J. E. McConnell promises suitable space for all exhibits.

Transportation: Dr. George Varina will

arrange for cars for the golfers and any others who request them. Please submit requests at the registration desk.

Barbecue: Dr. D. T. Milam and Dr. Henson Coon will see that everyone is well taken care of at the barbecue.

Decorations: Will be taken care of by the Ladies Auxiliary Committee—"nuff said."

Please write or telephone these chairmen if they can be of any service to you.

WILLIAM L. BENDEL, M. D., *Chairman, Publicity Commitee.*

WELCOME TO THE TWIN CITIES AND OUACHITA PARISH

Monroe and West Monroe, the Twin Cities of Northeast Louisiana are interesting and traditionally southern cities situated on opposite banks of the Ouachita River. These beautiful cities vividly contrast the old romantic south with the latest in modern community development. Homes, churches, schools, and business houses reflect the architecture of our times. Monroe and West Monore are constantly engaged in joint efforts to make this a better community in which to live.

Monroe's growth is the phenomenal story of a rural village shedding its swaddling clothes over night and growing into a modern metropolitan city within a decade with an estimated population in 1946 of 33,000.

West Monroe, which began to grow rather rapidly following the developments of gas fields had an estimated population of 12,000 in 1946.

The Twin Cities, 45,000 strong, are a staunch pillar in the economic, commercial, educational, religious, political, and social structure of Louisiana.

Selman Field operated during the war as a bombardier and navigation training center, is now leased to the City of Monroe as a municipal airport.

The Twin Cities are located on transcontinental highway 80 and five other highways radiate from these cities.

The five largest hotels have a total of about 800 rooms.

Northeast Jr. College of the Louisiana State University, located in the outskirts of the beautiful Sherrouse Addition, ranks as one of the leading institutions of the state.

Natural gas development began in 1916, grew in the next quarter century to the point where the "Monroe Gas Field" was the largest in the world.

Monroe and West Monroe have a commission council form of government. Monroe owns its public utilities and transportation facilities. The city is heated with natural gas. The Mayor of Monroe is H. H. Benoit; H. G. Bailey, Jr., Secretary of Chamber of Commerce. William Rodriguez and Ruffin Tidwell are Commissioners. In West Monroe, the Mayor is C. C. Bell.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

THE COUNCIL ON MEDICAL SERVICE AND PUBLIC RELATIONS NOW HAS THREE WEEKLY RADIO PROGRAMS

The Council on Medical Service and Public Relations now has three radio programs on an active basis. The programs are broadcast weekly and the radio stations use the AMA transcribed series and present them in cooperation with the Council on Medical Service and Public Relations of the Louisiana State Medical Society and the local par-

ish medical society.

The two new stations added to the broadcast schedule are KTBS, Shreveport, and KWCJ, Natchitoches. "The Story of Surgery" is being presented every Saturday at 4:00 p. m. over KTBS in Shreveport, and "Keeping Your Baby Well" may be heard over KWCJ in Natchitoches every Monday at 3:30 p. m. KNOE in Monroe, the first station to present our series, is broadcasting the program "What to Do Before the Doctor Comes," every Wednesday at 1:30 p. m.

All of the series are of a 13-week duration and when completed will be followed by other series.

Doctors in the listening areas should encourage their patients to listen to the broadcasts.

AMERICAN ACADEMY OF GENERAL PRACTICE OF LOUISIANA

Since this organization was formed last July, it has made unusual progress. The national organization lists about 3,300 members, with 45 states and one territory represented. Four states have organized on a state level, and many more contemplate organization in the near future. Kansas City will become the permanent home of the national organization next month. An executive secretary, Mr. Mac Cahal, probably the most capable person in his field, has been employed and is now gradually taking over the active management of the national organization. Plans for a journal are expected to be made within a year. Louisiana has been a leader in the movement from the beginning. Not only was this the first state to organize on a state level, but also all the congressional districts are organized on a district level. This will be the first state to hold a scientific meeting which will be held at the Bentley Hotel in Alexandria on April 3-4. Every doctor is cordially invited to attend. Watch your newspaper for further news about the meeting.

THIRD ANNUAL NATIONAL CONFERENCE ON RURAL HEALTH

Dr. F. S. Crockett and his committee on rural medical service of the A. M. A. presented one of the most interesting meetings held on this subject, at the Palmer House on February 6-7. The theme of the program this year was CHILD WELFARE.

About three hundred were present from all over the United States and represented outstanding medical men and laymen interested in rural health. Louisiana had ten representatives including eight doctors, Mrs. T. R. Tomlinson, chairman of the rural health council of Louisiana and Mr. Frank Lais, Jr., executive secretary of our Council on Medical Service and Public Relations. The general medical field was represented by both specialists and general practitioners.

The following items were almost completely agreed on by everyone present.

1. Rural areas are not as healthy as the urban centers.
2. Rural people, though obtaining the best medical care that they have ever had, still have poor medical care as compared to those in the city.
3. There is a definite need all over the country for better equipped hospitals in rural areas.
4. Rural areas need more and better trained general practitioners, dentists, and other allied professional help.
5. Many rural areas are badly in need of well located and well operated health centers.
6. Teachers in our educational system should be more health conscious and should have some extra training in health.
7. Better medical care is dependent upon improved economic standards in many localities. This is particularly true in the poorer districts.
8. Regular and complete physical and dental examinations should be a "must" in rural areas particularly among the school children. Many states are requiring that school children be examined every three or four years.

Probably the most fascinating part of the program was a symposium entitled "Rural Youth Looks at Health" given by four youngsters, representing the Grange, the Farm Bureau, the Farmers Union and the Cooperative Milk Producers Federation. One had just finished high school, two were still in college and one is a rural school teacher; all are probably still in their teens. They opened our eyes in many respects because we were surprised at their knowledge of conditions of rural health. They are demanding better rural health and they expect and will get better rural health.

The following four problems confront the farmer today according to their presentation.

1. Nutrition.
2. Poor health and poor educational facilities.
3. Few or no health units. Poor or no hospital facilities in the community.
4. The vanishing general practitioner.

In discussing nutrition they stated that the nutrition of the farmer was poor for two reasons. First, that he had not been educated to the fact of what is good nutrition and, second, that many times he sells off his best foods in order to improve his economic condition.

Poor health and education are caused by a number of situations; the poor economic condition of the farmer in many cases, the general tendency of everyone to ignore the farmer in preparing for good health and the indifference of the farmer himself many times to health problems. They stated that the lack of education and poor health go hand in hand.

While cities and urban centers have been very alert in putting in health units and good hospitals, rural areas have been very backward in carrying out these problems. It was brought out that this is as much the farmers' fault as it is the doctors'

but if we do not solve them, the politicians will ultimately take over. Only one of the four representatives advocated compulsory health insurance; the other three definitely recommended voluntary prepayment medical and hospital insurance as one of the answers to their problems.

Then they took a particularly hard swing at the general practitioner. They stated that the specialist had taken over but indicated that what the rural people need more than anything else to give them good medical care is a well trained general practitioner.

They are working on the above problems and expect to solve them.

Health education was stressed as an important factor in all school health programs. It was stressed that the teachers themselves should be better trained in health. The school lunch program in most cases should be enlarged and expanded to take care of all, but community cooperation is important to put these projects through.

Rural public health organizations such as our Rural Health Council in Louisiana is an important factor in looking after needs of the people in rural areas. The role of the Parent-Teachers Association, the health units and dental care was stressed. Woman's Auxiliary organizations can be called in many times to assist in putting through these public health programs.

Dr. Hoge from the Surgeon General's office of the United States Public Health Service stressed the importance of the Hill-Burton Bill to construct rural hospitals. With one-third of the total expense put up by the Federal Government and one-third by the state (if that is done) this leaves only one-third of the total construction to be borne by the community and makes it possible for many communities to have a well organized hospital which otherwise they would not be able to afford.

Dr. R. V. Platou, of Tulane, gave a remarkable paper on the pediatric service being carried out under the auspices of the State Board of Health and L. S. U. and Tulane Medical Schools. He stressed the role of the general practitioner in connection with health of children in rural areas.

Summarizing, it is realized as never before that rural young people want and are going to have good medical care. They are willing to work for this and expect our help and cooperation. Dr. Crockett and his committee, the rural medical services of of the various states and the farm people, through their own organizations, are all working cooperatively to give good medical care. None of these wants state or socialized medicine.

NEWS ITEM

Dr. L. Roland Young, formerly of Covington, Louisiana, is now practicing in Daytona Beach, Florida and recently received an appointment as member of the Consulting Staff of the Lake County Medical Center in Eustis, Florida. Dr. Young also recently addressed the Volusia County Society of the International Child Educational Association on "Mental or Psychological Prophylaxis in the Primary Grade Child" at the Lenox Avenue School, Daytona Beach.

EXPANSION OF A. M. A. SPEAKERS' PROJECT

The Board of Trustees of the A. M. A. has assigned a new duty to the Bureau of Health Education.

The bureau hereafter will endeavor to make more speaking engagements for traveling members of the A. M. A. headquarters staff.

The idea behind this assignment is that when a speaker travels a considerable distance from Chicago to attend a meeting or make an address, his time in the distant community will be used to the greatest possible extent. Most communities are eager for speakers from A. M. A. headquarters, and the Bureau of Health Education has been instructed by the Trustees to procure from all headquarters offices the names and speaking qualifications of traveling representatives from the A. M. A. The Bureau will then endeavor to arrange additional engagements in the community to which the speaker is going or in nearby communities.

The Bureau of Health Education desires particularly to have Women's Auxiliaries to local medical societies register their needs for speakers.

These requests will be coordinated with the availability of traveling members of the A. M. A. headquarters staff whenever possible, so as to furnish local communities with the service of these traveling representatives.

The Bureau will also be glad to hear from the secretaries of state and county medical societies the names of available speakers in their jurisdictions so that requests for speakers received at headquarters can be referred to local speakers, either direct or, if preferred, through the secretaries of state and county medical societies.

This experimental service, if successful, can be the nucleus for the nationwide Speakers' Bureau which, in coordination with local speakers' bureau organizations within the structure of the A. M. A., can render an important service to the public through the medical profession.

THE LOUISIANA-MISSISSIPPI OPTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

The Annual Meeting of the Louisiana-Mississippi Opthalmological and Otolarwngolocical Society will be held at the St. Charles Hotel in New Orleans on Saturday, April 17.

Program

8:30 a.m.	Registration
9:00	Call to Order
	Preliminary Announcements

	Appointment of Committees
	President's Address: Dr. Noel T. Simmonds, Alexandria, La.
	Secretional Obstruction and Bulbar Poliomyelitis, Dr. Thos. C. Galloway, Evanston, Ill.
	The Preparation of Patients for Intraocular Surgery, Dr. Walter Stevenson, Quincy, Ill.
12:30 p.m.	Luncheon
	Executive Session
2:00	The Management of Complications of Ocular Surgery, Dr. Watson Gailey, Bloomington, Ill.
	The J. Raymond Hume Memorial Address—Vertigo: Differential Diagnosis and Treatment, Dr. J. R. Lindsay, Chicago, Ill.
5:30	Cocktail Party for Fellows, guests and their ladies
	The Registration Fee of $10.00 includes the luncheon and the cocktail party.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY, INC.

The general oral and pathology examinations (Part II) for all candidates will be conducted in Washington, D. C., by the American Board of Obstetrics and Gynecology from Sunday, May 16, through Saturday, May 22, 1948. The Shoreham Hotel in Washington will be the headquarters. Formal notice of the exact time of each candidate's examination will be sent him several weeks in advance of the examination dates. Hotel reservations may be made by writing direct to the Shoreham Hotel.

Candidates in military service are requested to keep the Secretary's Office informed of any change in address.

Applications are now being and will be received until November 1, 1948 for the 1949 examinations. For further information and application blanks address Paul Titus, M. D., Secretary, 1015 Highland Building, Pittsburgh 6, Pennsylvania.

AMERICAN SOCIETY FOR THE STUDY OF STERILITY

The Fourth Annual National Session of the American Society for the Study of Sterility will be held in Chicago at the Congress Hotel, on June 21-22. The two-day program will be divided into a special series of panel discussions on male infertility, with papers to be read on female and miscellaneous infertility aspects, on the second day. Additional information concerning this meeting may be obtained from the Secretary, Dr. John O. Haman, 490 Post Street, San Francisco 2, California.

JOHN TOLSON O'FERRALL, JR.
1885-1948

Dr. John Tolson O'Ferrall, Jr., Councilor of the Second Congressional District, died in New Orleans on January 31, 1948. Dr. O'Ferrall, a graduate of Tulane University Medical School in 1908, was an active and interested member of organized medicine for many years. He served on many committees of the State Society and was Councilor of the Second Congressional District from 1943 until his death.

JOSEPH ALPHONSE O'HARA
1869-1948

Dr. Joseph Alphonse O'Hara, of New Orleans, died on February 25. Dr. O'Hara was president of the Louisiana State Medical Society 1938-39 and actively interested in the affairs of the organization at all times. He was a graduate of the 1900 Class of Medicine of Tulane University.

WOMAN'S AUXILIARY

At a meeting of the Ouachita Parish Auxiliary in January Mr. Frank Lais, Jr. spoke on Louisiana Physicians Service, Inc. and activities of the Council on Medical Service Public Relations of the State Society. Radio programs sponsored by the Louisiana State Medical Society and the Woman's Auxiliary of Ouachita Parish will be presented through the cooperation of KNOE as a public service feature. The topics of the first series of programs will be "What to Do Until the Doctor Comes". Mrs. H. V. Collins will serve as general chairman of a committee whose object will be to present the aims and ideas of the Louisiana Physicians Service to the public. Mrs. C. B. Flinn is radio chairman, Mrs. Hayden Cutler, public relations chairman and Mrs. J. W. McConnell and Mrs. C. P. Gray, Jr., will head the speakers' bureau.

Plans were discussed for the State Convention to be held in Monroe and capable Mrs. De Witt Milam was appointed chairman.

Here is the list of activities that are of importance to the Auxiliary.

Monday, April 12

12:00 noon—Luncheon honoring state and parish officers at the La Casa Tea Room, $1.25.

4-6 p. m.—Tea honoring Mrs. Warren, Mrs. Owens, Mrs. Anderson and the wives of the guest speakers, to be held at the home of Dr .and Mrs. A. G. McHenry, 1810 Riverside.

Tuesday, April 13

12:30 p. m.—Barbecue, Bernstein Park.

8:00 p. m.—Banquet and Dinner Dance, Hotel Frances.

Wednesday, April 14

12:30 p. m.—Style Show and Luncheon, Virginia Hotel, $2.00.

BOOK REVIEWS

Diseases of Metabolism: Edited by Garfield G. Duncan, M. D. 2d ed. Philadelphia, W. B. Saunders Company, 1947. Illus. Pp. 1045. Price, $12.00.

The second edition of this excellent book, completely revised and presented in a new slenderer format (though the text has actually been somewhat expanded) will be heartily welcomed by student, general practitioner, and internist alike.

The five years between the appearance of this edition and its distinguished predecessor have seen many significant advances in the field of metabolic diseases, and in no other text are these advances presented as conveniently and accessibly as here. In addition to the familiar chapters on carbohydrate, protein, lipid, mineral, water, and vitamin metabolism, all completely revised new chapters on thyroid and kidney disease have been added.

Dr. Duncan and his authoritative co-authors have produced a book which no internist or well-informed general practitioner can afford to be without.

J. R. SNAVELY, M. D.

Textbook of Human Physiology: By William F. Hamilton, Ph. D. Philadelphia, F. A. Davis Company, 1947. Pp. 504. Price $6.00.

Teachers of physiology to medical students have long felt the need of a smaller textbook in keeping with the length of time devoted to the formal teaching of this subject in the first or second year curriculum. Dr. Hamilton has attempted to supply this need by preparing a textbook in which the material is presented dogmatically and restricted to the things that the average student can encompass in the short time he is in the Department of Physiology. Thus the entire complex field is presented in about 480 pages.

For the most part, Dr. Hamilton has succeeded quite well in his difficult objective. The various sections on the nervous system, respiration, blood, circulation, digestion, fluid exchange, metabolism and the endocrines are well organized, adequately illustrated and the material clearly presented. By way of minor criticism, the question may be raised as to whether some of the material on methods of measurements of circulatory pulses, etc. might not have been abbreviated and the space given to some other topic, such as the endocrine system. This system is treated in 33 pages although the author in his introductory chapter emphasizes that "disturbances in endocrine function form an important chapter in modern medicine." The textbook will be chiefly valuable to the first year student who supplements his reading with the information obtained from lectures and who will go to the more detailed texts for special and additional information when necessary.

H. S. MAYERSON, PH. D.

The Physico-Chemical Mechanism of Nerve Activity: By David Nachmansohn, et al. Annals of the New York Academy of Sciences, *XLVII*, Art. 4, 1946. Pp. 375-602.

This monograph embodies the lectures given at a conference held under the auspices of the New York Academy of Sciences in which the leading investigators in the field participated. The lectures covered the most recent experiments in the field of nerve activity, synaptic transmission, and nerve metabolism. There are twelve lectures on various aspects of these fields, an introduction by Tracy J. Putnam, and a conclusion by John F. Fulton. Much of the work reported is concerned with the question as to whether the transmission of nerve impulses is electrically or chemically mediated. The electrical hypothesis is upheld by Dr. Eccles, whereas, Nachmansohn has given in detail his reasons for believing in the chemical mediation theory. Of particular interest is the lecture by Dr. Gerard entitled "A Critique of the Role of Acetylcholine," in which he questions the importance of acetylcholine in junctional transmission in the central nervous system and suggests that the time is ripe for a new concept of nerve impulse transmission.

The monograph is recommended to those who are interested in obtaining a first hand report of the modern concept of nerve activity as given by the workers who are best equipped to present this information.

H. S. MAYERSON, PH. D.

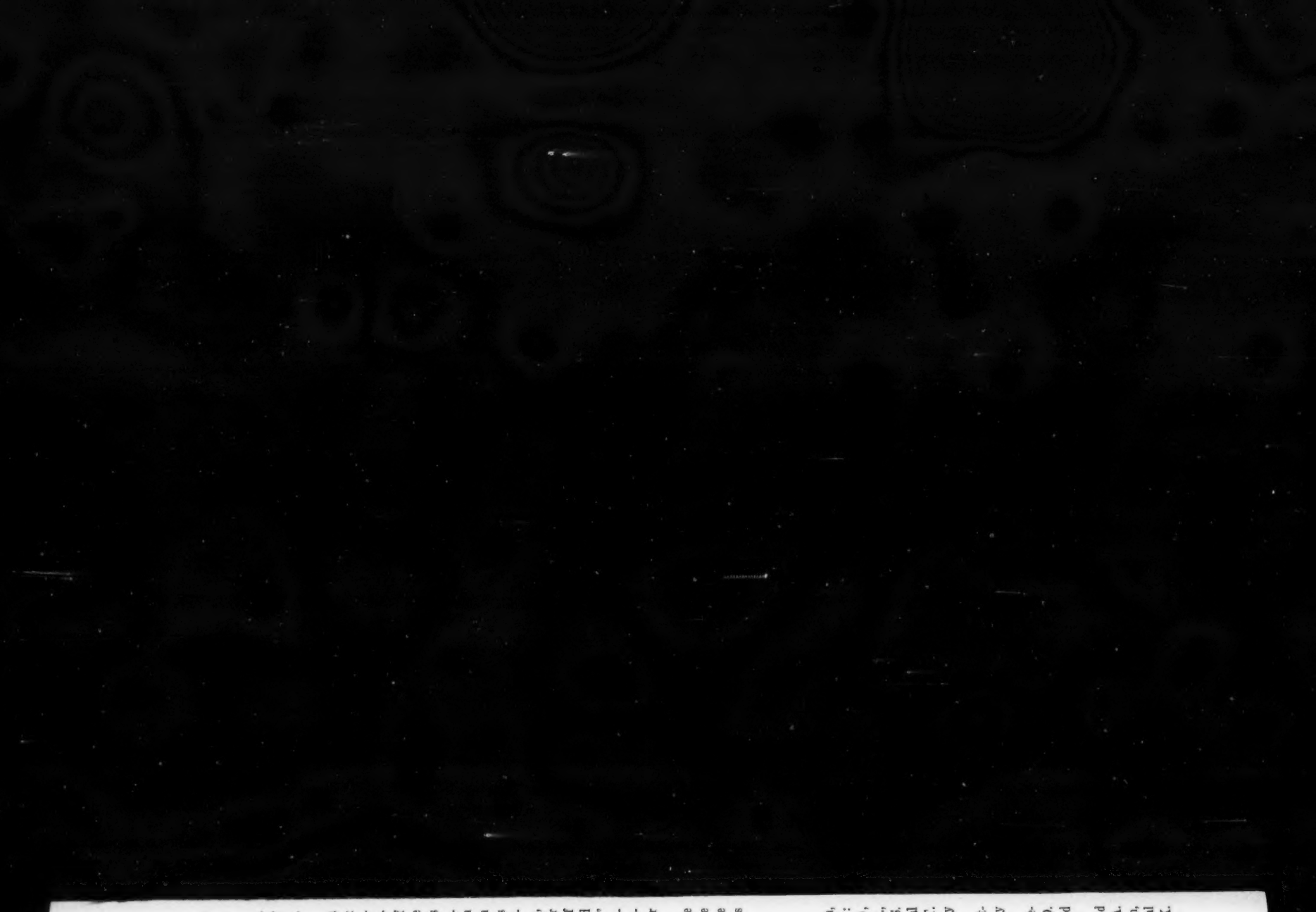
PUBLICATIONS RECEIVED

Butterworth & Co. (Publishers), Ltd., London, England and C. V. Mosby Company, St. Louis: British Surgical Practice (8 Volumes, Volume 1), edited by Sir Ernest Rock Carling, F. R. C. S., F. R. C. P. and J. Paterson Ross, M. S., F. R. C. S.

C. V. Mosby Company, St. Louis: Occupational Medicine and Industrial Hygiene, by Rutherford T. Johnstone, A. B., M. D.; Hernia (3rd edition), by Leigh F. Watson, M. D., F. I. C. S.

W. B. Saunders Company, Philadelphia: Treatment in General Practice (6th edition), by Harry Beckman, M. D.

Williams & Wilkins Company, Baltimore; Skeletal Tuberculosis, by Vincente Sanchis-Olmos, M. D.

The following publications were incorrectly listed in the January, 1948 issue:

Lea & Febiger, Philadelphia: The Foot and Ankle (3rd edition), by Philip Lewin, M. D., F. A. C. S.; A Primer of Cardiology, by George E. Burch, M. D., F. A. C. P. and Paul Reaser, M. D., Surgical Disorders of the Chest (2nd edition), by J. K. Donaldson, B. S., M. D., F. A. C. S.